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Psychiatry

Psychiatric Treatment Units in General Hospitals

George C. Sisler, M.D.

There have been emotionally disturbed patients in General Hospitals ever since the first hospital was built. Most of these patients suffer from the somatic accompaniments of fear, anxiety, or depression, but some have frank mental illness. However, adequate facilities for their treatment have been developed only recently—particularly since the Second World War. This development of General Hospital treatment facilities for psychiatric patients has been an outcome of the expansion of the private practice of psychiatry, and has been evidenced by the development of special psychiatric treatment units.

It is not a new concept to treat psychiatric patients as a group on specially equipped wards of General Hospitals. The first General Hospital in the United States, the Pennsylvania Hospital in Philadelphia, opened over two hundred years ago (in 1752) was originally established for the care of diseases of both the body and the mind. The building of this hospital was a Quaker enterprise, and supported particularly by that great American, Benjamin Franklin. The Act passed by the Assembly in this regard reads, "to encourage the establishment of a Hospital for the Relief of the Sick Poor of this Province, and for the Reception and Care of Lunaticks."[1]

The psychiatric unit of the Pennsylvania Hospital was in the basement, and the treatment given, we today regard as nonsensical and harmful —blistering, bleeding, purging, etc. This is of no importance. A hundred years hence our present-day treatment will be viewed in a similar light. The point is that cure, rather than cruel custody, was the underlying principle of treatment. The best medical knowledge of the day was used for the patient's benefit.

In the 1800's the care of the mentally ill passed from the general hospital to a vast number of Mental hospitals, mostly under State Government control.

The early years of this century saw the development of a new kind of treatment centre—the Psychopathic hospital. These had been in operation in Germany long before their introduction on this continent. Still separate mental institutions, they are designed to administer brief intensive treatment in incipient and acute cases. Usually located in large cities they are either designed as reception units for large mental hospitals, or are connected with a general hospital and a university. They provide educational facilities for undergraduate and graduate medical students, and have been centres for clinical and pathological research in nervous and mental disorders.

The Winnipeg Psychopathic Hospital, opened in 1919, was the first such unit in Canada, and has over the years kept pace with advances in the treatment of mental illness. This, and similar hospitals, have served to bring psychiatry closer to the rest of medicine, and have permitted most mentally ill patients to avoid going miles out in the country for treatment in large mental hospitals.

Despite the great advance they represented, in the years while Psycopathic Hospitals were the only in-patient treatment facility, two disadvantages were apparent. First this being the only adequately equipped hospital facility available, all mentally ill patients in need of hospital treatment had to be admitted by a legal procedure to a locked ward. Psychiatric patients and their families were faced with a choice between no treatment and admission to a hospital where disturbed and frankly psychotic patients of all types were treated. Often the result was that early treatment was not sought. Scondly, despite the expansion in the past ten years of the private practice of psychiatry, patients of these psychiatrists when ill to a degree necessitating hospitalization, could not be treated adequately by them, but were turned over to physicians of the psychopathic or mental hospital. The need obviously has been for the operation of adequate treatment facilities for psychiatric patients outside of provincial government hospitals. With the development in the past decade of new empirical therapies, particularly electroshock, the hospital treatment of the majority of mental diseases can be accomplished in a few weeks.

Now no one with any insight into the problem would consider attempting to treat all mentally ill patients on wards of general hospitals. There always have been, and in the foreseeable future will be, psychiatric patients who so lack insight that they must be forced to enter hospital, or who by the aggressiveness, noisiness, or self-destructiveness of their behavior necessitate confinement

[1] From a paper presented before the Winnipeg Medical Society, Winnipeg, December 10th, 1954.

by legal means behind locked doors in a government hospital. With modern methods of treatment most of these recover, but prior to and during treatment they are not suitable for general hospital care.

The point is that these are in the minority. Most persons with mental disease of neurotic or psychotic proportion, necessitating hospital treatment, have sufficient insight and are sufficiently controlled in their behaviour to be treated on a ward of a General Hospital. These depressed, anxious, fearful persons are thus spared the necessity of admission to psychopathic or mental hospitals. The providing of adequate facilities for their care in general hospitals is the thesis of this paper — the providing of facilities for their care and treatment as for any sick person.

The treatment of psychiatric patients in any numbers on medical wards of general hospitals in Winnipeg is a development of Post-War years. The establishment of special psychiatric treatment units is a development of the present and future.

Before outlining the set-up of such a unit perhaps one should deal with a common query. If we are moving in the direction of treating psychiatric patients like any other ill person, why not just admit them to various medical wards with the pneumonias and the leukemias. This is the case, of course, with those patients cared for by private psychiatrist in general hospitals where there are no separate psychiatric units. This arrangement has not worked out very satisfactorily because the mentally ill patient requires facilities for his care not available on medical wards. Though I would be the last to suggest that the psychiatric patient has a disease unrelated to that of the physically ill, his major treatment needs are different. This will be evident when we discuss the planning of a general hospital psychiatric unit.

In 1952[2] 10 Canadian hospitals had separate psychiatric treatment units. A report of Mental Health Services by the Department of National Health and Welfare in July, 1954, indicates that there are now such wards in 20 hospitals.

Though psychiatric patients have been and are treated in increasing numbers in General Hospitals in Winnipeg, no separate treatment unit was established until, in 1951, an in-patient service of about 10 beds was opened at St. Boniface. The present building plans of several of our hospitals call for the establishment of special psychiatric treatment units of anywhere from 20 to 30 beds each.

An adequate psychiatric department of this size can be housed in an ordinary hospital wing without prohibitive remodelling costs. A T-shaped floor plan is often found most suitable, though this is by no means essential. There is no need for separate units for men and women, and the present hospital planning trend of having private and public patients in one sub-specialty ward is a useful one so far as these units are concerned.

Last spring I had the opportunity of inspecting such wards in a number of mid-western American cities and in Toronto. Everyone is still learning how best to plan these facilities but certain general trends have emerged.

Single and two-bed rooms can be utilised, though if building costs permit, larger wards are best avoided. The belief that adult psychiatric patients do best in the group setting of large rooms does not stand practical test. Day room and occupational therapy space provides means of bringing patients together, but some need is felt at times during the day by all patients to have refuge from the crowd in a single or two-bed room. Patients are admitted voluntarily as to any hospital bed, and doors are not locked. The name of the ward should be in keeping with the system used for identifying other wards. Special unbreakable window screens are usually installed, although it would seem that these are not essential, particularly if the ward is on the ground floor. I have seen new psychiatric units in Toronto and Rochester, N.Y. on the third and sixth floors. These had ordinary window screens and the only safety device was that the windows had locks that prevent their being raised more than 12 inches. Patient suicide has not been a problem.

Special attempts to make the ward "totally secure" tend only to defeat the purpose of the plan. Tiled walls, built because of a fear that irresponsible patients will soil or damage them, heavy furniture, furniture bolted to the floors, no curtains, heavy window screens, no plants, caged-in nurses' stations — all these are unnecessary. The reports I got from staff in units I visited was that if the ward was nicely decorated and pleasant, ordinary social convention prevented damage being done. The tiled walls only seemed to be license for aggression and unrestraint.

It takes a pretty enlightened architect to plan such a unit, for he likely has in the past planned psychiatric wards only for mental hospitals. What is wanted in a general hospital is quite different.

What may be called the "fire extinguisher problem" illustrates this point. In planning some of these units the fire-inspectors said that extinguishers have been available to everyone, yet the hospital administration wanted them locked up, envisaging patients squirting them wildly about. In practice this is never a problem. Big red fire extinguishers and hoses sit out in the hallways for years and are never touched. The fire extinguisher is a problem in internes' quarters, not on psychiatric wards.

Of course, from time to time a patient will become suddenly and unexpectedly disturbed. With a proper admission policy and adequately

trained nurses, however, this happens on the psychiatric unit only slightly more frequently than on other hospital wards.

On each unit about two special secure rooms are necessary. These are sound-proofed, have locked doors and special window screens, and should be air-conditioned. Though the policy is not to admit patients so disturbed that they require care in such rooms, it occasionally happens that a patient while on the ward, or indeed in another hospital ward, becomes suddenly disturbed and temporary care in seclusion is needed in anticipation of treatment or transfer to a Psychopathic Hospital.

Since most patients are ambulant, rooms must be provided for occupational therapy and recreation. In many of these units the day sitting-room is used as a common dining area.

Early in the development of these wards, some persons found it difficult to accept the idea of providing space and personnel for recreational and occupational activities. The reasoning seemed to be that anyone able to be up and about most of the day and to engage in these activities probably did not need to be in hospital in the first place. But these group activities are a very necessary part of rehabilitation which, with most patients, starts from the day of admission. Regression into the apathy and dream world of mental illness is fostered by nothing so much as just lying in bed or sitting in a chair.

These facilities for diversion for ambulant patients have led to the development, particularly in Montreal, of what are called **day treatment units**[3] either on the general hospital in-patient psychiatric ward, or in separate areas. These **day treatment units** offer something halfway between in-patient and out-patient care. It consists of 8 hours daily of hospital treatment for out-patients. This allows the treatment of numerous persons for whom, because of their other responsibilities, it would otherwise be impossible. Some of these patients who spend the day in hospital, and the evening and night at home, have electroshock or insulin treatment in the morning, and psychotherapy or occupational therapy in the afternoon.

On psychiatric units many non-patient rooms of the treatment and service variety are needed—room for insulin therapy, electroshock, interviewing and psychological testing, as well as the occupational therapy and recreation rooms I have mentioned. The initial construction costs are higher than for other wards. The cost of maintaining the ward too, is increased since there are a smaller percentage of the rooms occupied by patients, and more personnel are needed. These justify patient rates somewhat higher than in other parts of the hospital.

Increasingly Blue Cross plans are covering the treatment of mental illness in general hospitals. A Survey[4] in 1953 of the 81 American Blue Cross plans shows that:

22% — offer coverage on the same basis as other illnesses.

48% — offer partial coverage; but

30% — still exclude mental disease.

Our Blue Cross plan at present falls in the "partial coverage" group, offering care only for patients receiving electroshock or insulin. A limit is imposed of 21 days for each admission for electroshock and 12 days for insulin therapy. As well the total days permissible in the lifetime of the contract are limited.

The general hospital psychiatric service provides facilities for the treatment of patients admitted for psychiatric disorders, and also for those patients, who in the course of hospitalization for another reason, develop a psychiatric illness. Mutual consultation with other specialists is facilitated.

The importance of these units in undergraduate and graduate psychiatric medical teaching and in nurses' training is apparent.

The matter of personnel has been mentioned only in passing, but the selection of suitable nurses and attendants is of the utmost importance. It seems that some persons with sympathy, interest in patients, and tact, are able to function well on psychiatric units. Others cannot, they are impatient, rejecting of the ill person, and mutual antagonism develops. Finally, it is obviously necessary that for public cases, an out-patient psychiatric service be provided for former patients and for those for whom hospitalization can be avoided. These out-patient services in Winnipeg have developed far ahead of in-patient facilities, and are a topic for discussion in themselves.

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Medicine

Manifestations of Liver Failure

J. H. Martin, M.D.

When looking at a cross section of normal liver tissue under the microscope, the similarity of the appearance of all the parenchymal cells is impressive. The number of functions ascribed to these cells is multitudinous and ever expanding. We can only assume that each cell takes part in all these functions and that there is no division of labor. What happens, when through chronic disease, the liver fails to perform these multiple duties? Do they all fail simultaneously; or is it simply a numerical reduction of functioning units which become over taxed; or can a single cell lose its ability to perform certain tasks and still competently carry out others? We are beginning to see some of the answers by the means of histochemistry, but much is still to be learned. Several books contain very orderly charts of the sequence in which functions disappear, but often the clinical case won't fit the chart.

At the outset of this paper, I thought it would be a neat exercise to line up the more important functions attributed to the liver and then join them with the clinical manifestations which their loss produced. It soon became apparent that about even the best recognized symptoms and signs too little was known to make this profitable. I can only claim that the following features can maintain some continuity because of their basic theme of disturbed liver function. Jaundice, portal hypertension, and ascites are complete subjects in themselves and will not be discussed. Hepatomegaly, while not essential, directs our attention to the possibility of disease in the organ. Yet not all palpable livers are diseased.

I. Non-specific Symptoms

Many symptoms have been attributed, in the past, to disturbed liver function without any real basis. These would include such things as indigestion, nausea, constipation, diarrhoea, torpor and just feeling plain "liverish". While these may be true symptoms, they are too non-specific to be of use in diagnosis.

II. Pruritus

This is very common, except in uncomplicated hemolytic jaundice. It is often said that it is the result of irritation of sensory nerve ends by bile salts deposited in the skin. Actually, its mechanism of production is quite unknown. Certainly it is not produced nor aggravated by injection of either bilirubin or bile salts. However, its occurrence is believed to be of good prognosis in parenchymal disease presumably because the liver has maintained its ability to produce bile salts. Also, it frequently disappears in the terminal state. It is said to be more severe in malignant than in benign obstruction but this is only relatively true. However, pre-icteric itching is strongly suggestive of a neoplasm. It is increased by augmented skin flow, being worse under bedclothes and is reduced by the vasoconstrictor ergotamine. Diminished production of choline esterase, which occurs in liver failure, has been held important; but of this there is little proof.

III. Anorexia and Weight Loss

The reason for appetite loss in the case of the diseased liver is as confused as the reason for its appearance in the healthy. Severe and absolute anorexia at the onset of infectious hepatitis is probably due to an enteritis produced as the virus transverses the mucosal barrier; but as to its mechanism in chronic liver failure, we are completely in the dark.

IV. Muscular Weakness and Asthenia

This common symptom occurs independently of weight loss and anorexia. It may be profound. It is characteristic of parenchymal disease and does not occur in obstructive jaundice. Several hypotheses such as low serum esterases, diminished vitamin E absorption, bile salt retention, delayed lactic acid reconversion to glycogen, and hypokalemia have been advocated, but none stand too careful scrutiny.

V. Pain

Why slow, placid, liver necrosis should at times be painful is difficult to envisage. Still, aching in the right hypochondrium in liver cirrhosis is not uncommon. Perhaps there are areas of sub-acute necrosis with inflammatory changes in the contiguous capsule that escape attention. Many of the bizarre diffuse abdominal pains may be due to abnormal vascular distension associated with the shifts in flow that accompany portal hypertension.

VI. Fever

Fever may result from actual liver necrosis, or an associated cholangitis, but it must be remembered that like nephrotic, those with liver failure are prone to cryptic low grade infections. This may be related to failure of production of certain immune proteins.

VII. Liver Breath (Fetor hepaticus)

The nature of this interesting and long recognized sign is still unknown. This sweetish pungent odor may be recalled by sniffing a portion from the butcher that you forgot to put in the refrigerator. In hepatic failure, it can be detected in the urine if it is allowed to stand in a shallow dish. It occasionally occurs in the breath and

From the Gastro-Intestinal Section, Clinical Research Unit, St. Boniface Hospital.

urine of perfectly healthy individuals. Its chemical composition has not been elucidated and it is not picked up by mass spectrophotometry. In general, it occurs only in advanced intra-hepatic disease but: (1) it sometimes occurs in obstructive jaundice, (2) it does not always appear before liver death, (3) it is not necessarily a sign of impending coma nor does it have anything to do with its production, (4) it is said to disappear with massive vitamin B complex therapy.

VIII. Steatorrhea

In lists of the etiology of steatorrhea, liver disease is always included. But as to just why this should occur is far from clear. It requires almost total exclusion of bile from the intestine before emulsification and absorption are interfered with, when from 25 - 75% of ingested fat is lost in the feces. Yet steatorrhea does occur. There may also be malabsorption of fat-soluble vitamins. This is evidenced by nyctalopia due to faulty absorption (or metabolism) of vitamin A, or bleeding due to lowered prothrombin from lack of vitamin K.

IX. Endocrine Changes

Estrogens are largely metabolized in the liver, being oxidized or conjugated with glucuronic or sulphuric acids. In hepatic insufficiency, in males, signs of hyperestrogen activity may be evidenced by gynecomastia and feminizing of the skin. Pubic and axillary hair may be lost.

X. Cutaneous Lesions

a. Arterial Spider. As their descriptive term implies, these consist of a central body with legs. The body may be slightly elevated or the legs only a diffuse erythema. When compressed under a glass slide, it can be seen that they are pulsatile and show the fiery redness of arterial blood which flows in a centrifugal direction. The statement that they appear in the area drained by the superior vena cava is almost true. They do occur in other locations, but only rarely. The reason for this distribution is unknown. They appear on visible mucous membranes and so probably also on those not seen and may be the source of hemorrhage. Again their mechanism or reason for formation is not clear. They appear during pregnancy but disappear shortly before term. For this reason they have been held to be related to blood estrogen levels. However, some apparently perfectly healthy individuals show these phenomena. They should not be confused with "venous stars", telangiectases of Osler's disease, or the familiar Campbell de Morgan spots which do not pulsate.

b. Liver Palms. A cherry redness of the hypothenar eminence, the base of the thenar eminence, the tips of the fingers and even occasionally of the feet has long been attributed to liver disease. Yet this is also seen in "normal" individuals. Bean, who has written much about liver palms, relates them to excess estrogens or possibly a disturbance of the glomus mechanism.

XII. Hepatic Coma

This rather unusual phenomena is held to be precipitated in those with liver disease by hemorrhage, surgery, infection, alcohol, narcotics and sedatives, high protein intake (meat intoxication syndrome), or paracentesis. Adams and Foley have described the main signs as those of mental apathy or sometimes delirium, a flapping tremor which may be brought out by extending the arms, and characteristic E.E.G. changes. Later patients lapse into an anaesthesia-like state in which various abnormal neurological signs may be found. While it is of serious prognosis, recovery often occurs. Its nature is still uncertain. There is no elevation of C.S.F. pressure, though the protein may be raised. Blood ammonia, which does not exist as such, and is difficult to measure, is often but not always elevated and is not correlated with the degree or presence of coma, organic acids and blood pyruvates are often elevated with low serum sodium, potassium, and magnesium, but these are non-specific changes. Walshe has directed attention to the interesting substance glutamic acid and has used it as therapy with variable results. Glutamic acid is the only amino-acid which will support brain respiration in vitro. It is broken down to a a-ketoglutaric acid which enters the citric acid cycle. It combines with ammonia and binds it to form glutamine. If the change of glutamic acid to glutamine is prevented, intracellular ammonia rises. He has also found an increase in methionine sulfoxide in the C.S.F. in coma. This is a known glutamic acid inhibitor. However, Bollman contends that this is not a preformed methionine sulfoxide but an artifact of oxidation.

XIII. The Hepato-renal Syndrome

One hesitates to mention this ill-defined and controversial subject. Yet, some individuals with advanced liver disease do become oliguric or anuric out of proportion to their degree of dehydration. It has been held that the kidney is poisoned by circulating toxins which have not been conjugated in the liver, and the pathological renal changes do resemble the "lower nephron" picture. However, much clarification is necessary.

It is hoped that this rather brief enumeration of symptoms and signs will point out the diversity of systems which may be involved by failure of this organ which is the focal point of so many metabolic processes. Many of the biochemical derangements are far from worked out. Still, we are beginning to see some of the correlations between the complicated formulae of the laboratory and the clinical picture in the living patient.

Neurosurgery

Non Traumatic Surgical Problems

Dwight Parkinson, M.D.

This talk has to do with some of the more common non traumatic lesions of the central nervous system amenable to surgical treatment. The following subjects will be briefly discussed: congenital malformations, intracranial tumors, intraspinal tumors, cerebro-vascular accidents and intractable pain.

Congenital Malformations

The hydrocephalics are probably the most common congenital problem coming to the attention of the neurosurgeon. The type and extent of hydrocephalus must be determined either by air or dye studies, and in some very few instances surgery may be of help, either by relieving a block with one of the various shunt procedures, or by relieving the compression of a herniated cerebellum and medulla as in an Arnold Chiari malformation. All too often, however, the hydrocephalus is merely part of a generalized defect in the development and function of the central nervous system, and receiving the fluid blockage does not result in a normal child. These are extremely difficult cases from all points of view because of the tremendous emotional factors on the part of the parents. When an offspring is found to be defective the parents feel a certain amount of guilt, and it is human nature to seek a place to put the blame. The surgeon must assume, if he operates, that anything short of a normal child will be dated back to his operation.

The encephaloceles should always be removed surgically and the defect repaired if the child appears to have an otherwise normal central nervous system. It should be mentioned that encephaloceles may present as nasal polyps or even as seemingly unrelated masses about the head and face, which may completely surprise an unsuspecting surgeon if he sends the tissue to pathology and gets a report back of brain tissue or even of brain neoplasm. Sometimes these encephaloceles become detached from their attenuated connection to the brain and present merely as a separate mass of brain tissue about the face or in the case of the myeloceles along the spinal column.

The dermoids and dermal sinuses deserve special mention. They are rare but because of their rarity frequently cause the practitioner trouble. One should remember to always be suspicious of any sinus about the head or along the spinal column in the very young. Similarly any mass near the midline over the neuroaxis which resembles a wen should be regarded with suspicion. It is wise to at least perform a pre-operative x-ray before attacking such a lesion surgically. They very often extend through the bone with a narrow neck connecting with an intracranial or intraspinal mass, which may be many times larger or which may in turn extend on to deep within the brain substance often ending in a ventricle.

The meningoceles and meningomyeloceles should only be dealt with surgically if there is normal or near normal function of the lower extremities and reasonable evidence that hydrocephalus is not also present, or if present, can be controlled. It must be emphasized that repair of the defect does not improve the neurologic deficit.

Brain Tumors

Brain tumors along with brain abcesses and subdural hematomas all have one feature in common; they occupy space and progressively enlarge. For this reason their signs and symptoms are all essentially the same, differing only with the location of the mass and in rate of progression. For practical consideration they are grouped together as, "space occupying lesion". The type of tumor is frequently never determined until its actual removal.

The correct diagnosis of a brain tumor begins with two coincident situations. First you must have a patient who has a brain tumor, and secondly the examiner must be a doctor who recognizes the signs and symptoms and becomes suspicious of the tumor. The first situation is not too rare. About half as many patients walk into your office each year harboring a brain tumor as with bronchogenic carcinoma. The second situation is, of course, less rare.

The path is fairly direct from this first suspicion to the conclusive diagnosis, but lacking this suspicion, the journey is never begun. In other words the suspicion of the first doctor visited is the most important feature in the diagnosis of brain tumor.

When does one become suspicious? Brain tumors manifest themselves in two basic fashions. Firstly by distorting the vessels and structures that surround and support the brain. This results in headache, the most frequent single symptom of a brain tumor. Secondly by distorting the nervous tissue itself. This results in seizures, hallucinations, mental and personality changes or neurologic deficits such as weakness, spasticity, aphasia, visual disturbances, ataxia, etc. With any one of these one should entertain the possibility of a brain tumor. With one of these plus headache one must be extremely suspicious.

* Read at the General Practitioners' Association Meeting on February 16, 1955.

The headache of an expanding lesion is fairly constant and over a period of time gets progressively worse. It may be accompanied by vomiting and is frequently worse in the morning; it may even awaken the patient before his normal awakening time. It is characteristically aggravated by strain, movement and changes in position, but not usually aggravated by noise and excitement. It is relieved by analgesics more than sedatives.

Seizures and, to a lesser extent, hallucinations must be considered due to a brain tumor until proven otherwise. This is especially true of seizures starting in adult life and more especially true with focal seizures. Long duration, even 20 years, does not exclude a tumor.

Mental and personality changes, of course, occur in many conditions other than tumor. However, unless these changes can be definitely attributed to some other condition, one is obliged to exclude a brain tumor in such a patient.

Neurologic deficits must be automatically compatible with an intracranial localization to arouse suspicion of a brain tumor.

There is no substitute for a careful history, physical and neurological examination. Funduscopic examination must always be done. The presence of papilloedema indicates increased pressure with rare exceptions. Its absence, however, does not exclude increased pressure. The visual fields are often a tremendous help in localizing a lesion.

When the doctor has progressed this far and is suspicious of a brain tumor he must then prove or disprove its presence. In this process localization is an automatic accompaniment. Often the lesion is localized accurately from the history and examination alone, but one must still prove that the lesion is occupying space. Skull and chest films should be the next steps in order. The latter because over 10% of all brain tumors are metastatic. Skull films may reveal local erosion, local vascularity, displacement of the calcified pineal or choroid plexus, osteomatous formation, erosion of the sella or clinoids, suture separation or even calcium in the tumour itself. Normal skull films do not exclude the possibility of a brain tumor.

If a lumbar puncture is deemed necessary, the pressure should be carefully measured with the patient horizontal, relaxed and his neck straight, not flexed. Jugular compression is contraindicated. Manometric measurement is the only reliable method of measuring pressure. Estimation by the rate of flow is completely worthless. One can have a rapid flow with a low pressure or a very slow drip from the needle with a pressure over 600. The fluid should be examined for cells, protein, Wasserman and colloidal gold as a minimal examination. On some occasions other examinations may be indicated. It must be remembered that a low sugar count and a high cell count may both be found with highly malignant tumors as well as with inflammatory processes.

From here one moves to specialized procedures with more specific diagnostic significance. The electroencephalogram may or may not localize an existing tumor. It will usually give evidence of abnormality in presence of a tumor but not invariably so. One is never justified in operating on the basis of an electrocephalogram localization alone.

Radioactive dyes in combination with modified Geiger counters and echo phenomena from ultrasonic waves used in much the same manner as radar have both been given considerable trial, but are not yet considered universally reliable.

The most commonly used, and, so far, the most reliable special procedures are the pneumograms, (encephalogram and ventriculogram) and the angiogram. Each has its peculiar advantages, disadvantages and dangers. The choice of procedure in each case is the neurosurgeon's. If these tests are negative one is forced to abandon a diagnosis of brain tumor at least for the time being.

The question always arises — when is it justifiable to submit a patient to these procedures. The answer: Whenever there is reasonable suspicion of a brain tumor, and the corollary is that negative results do not permanently exclude a brain tumour, but rather as with a negative barium series, the tests should be repeated after an interval, if the indication persists.

In summary, the diagnosis of a brain tumor begins with and is largely dependent on the acuity and suspicion of the first doctor visited. From here the history and examination, x-rays and special diagnostic procedures follow in logical sequence culminating in the definitive diagnosis and accurate localization or the exclusion of a brain tumor.

Spinal Cord Tumors

Most spinal cord tumors are benign and amenable to complete surgical removal with complete recovery if they are diagnosed in time. Cord tumors are invariably misdiagnosed by the first several physicians who see them. By the time they are actually recognized they have followed one or two courses. They have either had multiple abdominal operations for pain and/or they have been diagnosed as disseminated sclerosis. The extramedullary tumours are characteristically productive of pain at the level of the tumor with radiation around to the front following the nerve distribution of that particular level. This pain is somewhat intermittent and often worse at night. The pain mechanism is similar to that of a protruded disc, and the aggravating features are similar. Inasmuch as the majority of spinal cord tumors are in the midthoracic region most of the pain radiation will be to the upper abdomen and

lower thoracic region. The symptom of pain is followed or preceded by the gradual and often intermittent development of weakness, anaesthesia, paresthesias, and ataxia below the level. It is this development that accounts for the frequent diagnosis of disseminated sclerosis, particularly when the cord tumor is situated high. The warning cannot be repeated too often; never diagnose disseminated sclerosis without absolute exclusion of any other lesion that could produce the picture at hand. The intramedullary tumors are less characterized by pain at least until very late in their course. They do characteristically have a disturbance of pain and temperature sensation at the level of the tumor and a progressive loss of function from that level on down as the tumor enlarges.

The final diagnosis of a spinal cord tumor depends on myelography. This should always be associated with examination of the spinal fluid again for cells, protein, Wasserman and mastic.

Cerebro-Vascular Accidents

The cerebro vascular accidents for the most part are not amenable to surgical treatment. However a small percentage are curable by surgery. Probably nowhere does a physician feel so helpless and small in the imploring eyes of the waiting relatives as when confronted with a patient hemiplegic or unconscious or both who was a few minutes before a healthy active citizen. As with any problem one must approach the situation in his own mind with an orderly classification of the variants. No problem of such complexity is classified as simply as a cerebro-vascular accident. There are but two main types of lesion — those due to infarctions and those due to hemorrhage. The infarctive lesions are definitely not surgical problems. The hemorrhagic ones only on occasion are surgical problems. The hemorrhage may be either intracerebral or subarachnoid. Both may occur together as an intracerebral hemorrhage, may rupture into a ventricle or through the cortex. Conversely a primary subarachnoid hemorrhage may burrow deeply into the cerebral substance.

There is no certain clinical differentiation between a cerebral infarction and an intracerebral hemorrhage. Either may be steadily or intermittently progressive or may be abrupt. Either may be massive or slight; either may come on in sleep or in agitation; either may show fairly rapid improvement. The presence of gross blood in the spinal fluid will often settle the differential. There is a group of very definite syndromes which, if recognized, can be labelled with certainty as thrombosis. These are the syndromes of the brain stem arteries of which the posterior inferior cerebellar is by far the most common. These syndromes are in standard texts of neurology and need not be repeated here.

The rapid fulminating intracerebral hemorrhages offer little opportunity for therapy of any type. In those surviving long enough, attempts have been made to evacuate the clot and stop the bleeding with occasional success. This probably will be done more frequently in the future. It must be remembered however that whatever cerebral tissue is damaged by the hemorrhage and whatever is destroyed by the surgeon for exposure will remain destroyed.

The less vigorous hemorrhages offer more opportunity for relief. These usually stop spontaneously and the residual clot may behave much as a tumor. Removal of the clot may give considerable relief.

Spontaneous subarachnoid hemorrhage in over 90% of the cases is due to a ruptured intracranial aneurysm. These devastating hemorrhages usually occur in young people, occasionally preceded by minor warnings of simple headache resembling a migraine.

This condition offers a fascinating surgical challenge. There is no lesion more benign and yet more lethal than a leaking congenital intracranial aneurysm. The mortality rate is exceedingly high. From one third to one half die in their first attack. About the same proportion of the survivors will succumb in the well nigh inevitable following attacks. Many of the final survivors are neurologic cripples. The surgical mortality rate in various centres is considerably better than this although still quite high. A recent follow-up study was performed on the author's series ranging from 1 - 5 years. There were 38 cases of proven aneurysms who had survived their initial attack and were in an age group where surgery might be considered. Eight of these patients refused operation and of these eight seven are now dead. The remaining 30 were operated with a direct intracranial attack with four deaths. The mortality rate in the operative cases is thus 16% and in the non-operative cases 87%.

The proper handling of a spontaneous subarachnoid hemorrhage case is fairly standardized. First the diagnosis is established from the history of a sudden violent headache which may shortly progress on down the back of the neck and even into the lower spine and legs. This may then be followed with nausea, vomiting, collapse and even loss of consciousness. On examination the neck is usually rigid. There may be multiple hemorrhages visible by funduscopic examination. The spinal fluid if examined is diffusely red or pink and under increased pressure. Having established that the patient has a spontaneous subarachnoid hemorrhage he is kept well sedated at bed rest using laxatives to avoid straining until such time

as it is deemed advisable to perform an angiogram. This is quite safely done very early in the course and immediately clears up one large problem, namely, is the case operable or non-operable. If an aneurysm is visualized it is reasonable to assume that the bleeding came from that aneurysm and it is also reasonable to assume that it is going to bleed again repeatedly until it finally takes the person's life. Interestingly enough cases in which an aneurysm is not visualized are nowhere near as likely to have repeat hemorrhages.

Having localized the lesion and decided that it is operable the patient is still kept at bed rest until the optimal time for surgery. The longer one can wait the better the exposure because of the absorption of blood and the subsidence of swelling. However if one waits much beyond the second week the risk of a repeat hemorrhage becomes increasingly great. Most repeat hemorrhages from aneurysms occur in the second and third week. Hence the operation is probably best performed around the tenth post-hemorrhage day.

The ultimate aim in surgery is to isolate the aneurysm from the circulation without disturbing the remaining flow to the brain. This is done in a variety of means depending on the location of the aneurysm, its neck and its relationship to the circle of Willis.

The arteriovenous fistulas, although extremely formidable in appearance are actually less lethal to operate than the saccular aneurysms. There were five such lesions in the author's series, all of which were treated with block excision at a mortality rate of 0.

In summary, spontaneous subarachnoid hemorrhage must always be considered a surgical problem until proven otherwise. The mortality rate with a proven aneurysm is exceedingly high if left alone whereas in capable hands it is less than 20% if operated.

Intractable Pain

Intractable pain cases are often referred to the neurosurgeon for whatever help he has to offer. There are many procedures which can be done to relieve pain: section of a nerve, section of a posterior root, section of the pain track within the spinal cord or medulla, depending on the level of the pain. The pre-frontal leukotomy occasionally is of help in the relief of the apprehension and anxiety associated with pain. One axiom should always be remembered when dealing with pain and its surgical relief. That is, the sacrifice of a normal pain pathway is never justified in an undiagnosed condition. It is rarely justified in any condition other than malignancy. The exceptions are the major neuralgias of the face which are definitely amenable to surgical treatment. This includes however only the glossopharyngeal neuralgias and the trigeminal neuralgias. The other so-called neuralgias and the migraine headaches are not permanently relieved by any form of surgery although almost any type of surgical attack locally will give relief for a short period of time. As with phantom limb causalgia, post-operative scar pains, etc., the biggest mistake the surgeon makes is ever starting on the path which leads to multiple surgical operations in a futile attempt at permanent relief.

Obstetrics & Gynecology

The Treatment of Genital Prolapse

W. L. G. Quinlivan, M.B. M.R.C.O.G.
Resident in Obstetrics, Grace Hospital, Winnipeg

A doctor's training is based on a sound knowledge of Anatomy. There is probably no better example of the practical application of this knowledge than in the treatment of Genital Prolapse.

Anatomy of the Pelvic Supports

The anatomy of the Pelvic Floor has been described in detail in a recent edition of the Manitoba Medical Review (Hughes 1955). For the purposes of this article I would like to remind readers of the following points:

1. Ligaments

1. The Cardinal Ligaments are fan shaped structures, which arise from the pelvic walls and are inserted into the Supra Vaginal Cervix and upper third of the Vagina.

2. The Utero Sacral Ligaments arise from the third Sacral Vertebra and are inserted into the Supra Vaginal Cervix. They are continuous on either side with the posterior portion of the corresponding Cardinal Ligament, and are considered to be thickenings in the posterior borders of the Cardinal Ligaments.

3. The Sub-Vesical Ligament is a sheet of fascia and smooth muscle which extends from the posterior surface of the Pubis to the Supra Vaginal Cervix.

These ligaments are composed of Pelvic connective tissue supported by unstriped muscle. The Cardinal Ligaments are undoubtedly the strongest.

2. Levator ani Muscle

The Levators Ani consist of three pairs of muscles—Pubo-coccygei, Ilio-coccygei and Ischio-coccygei.

The Pubo-coccygei arise from the posterior border of the Pubis.

The Medial fibres (Pubo-vaginalis) pass backward, lie on either side of the lower third of the Vagina, and interdigitate in the Perineal Body. The Perineal Body lies between Vagina and Rectum.

The Central fibres (Pubo-rectalis) pass backward, lie on either side of the Rectum, and interdigitate between Rectum and Coccyx, in the Ano-coccygeal raphe.

The Lateral fibres (Pubo-coccygeus) pass directly back to be inserted into the Coccyx.

The Pubo-coccygei, by their interdigitations in the Perineal Body, support the Posterior Vaginal Wall, and prevent the lower part of the Rectum prolapsing forward into the Vagina.

The Ilio-coccygei arise from the White Tendinous Line on the Obturator Internus muscle, and are inserted into the Coccyx. They form the main muscular components of the Pelvic Diaphragm.

The Ischio-coccygei pass from the Ischial Spines to the Coccyx. They are the tail wagging muscles of the dog. In the human they merely form the posterior borders of the Pelvic Diaphragm.

3. Urogenital Diaphragm

This consists of two triangular layers of fascia superficial to the Levator Ani. Two sides of the triangle are attached to the Ischio-pubic rami. The third side is represented by a line joining the two Ischial Tuberosities. On this line the two layers of fascia fuse and become continuous with the Anal fascia.

Between the two layers of fascia lie the Sphincter Urethra and Deep Transverse Perineal muscles.

The two layers of fascia are pierced by the Urethra and the Vagina. They are firmly attached to these canals and sling them to the Ischio-pubic rami.

The Perineal Body lies in the midline immediately anterior to the line of fascial fusion. Both layers of fascia and the Deep Transverse Perineal muscles, are inserted into it. From within out it consists of — Levator Ani, fascia, Deep Transverse Perineal muscle, fascia and Superficial Muscles of the Perineum.

4. Superficial Muscles of the Perineum

These muscles lie superficial to the Urogenital Diaphragm. The Superficial Perineal, Bulbo-Cavernosus and External Anal Sphincter muscles, are inserted into the Perineal Body.

The Pelvic Organs in Relation to their Supports

The structures mentioned above form the main supports of the pelvic organs. They can now be considered in relation to the tissues they support.

The Body of the Uterus is supported by the Cervix.

The Cervix Uteri is slung to the pelvic walls by the Cardinal and Utero-Sacral ligaments.

The Vagina is surrounded by a layer of fascia. In its lower third this fascia is adherent to the Urogenital Diaphragm and the Levator Ani. The lower third thus has strong fibro-muscular attachments, anteriorly, laterally and posteriorly, to the pelvic walls and Perineal Body.

The Upper Vagina is attached to the Cervix. The Vaginal fascia is continuous with the Parametrial fascia. The Vagina thus has strong attachments to the Cervix Uteri. These attachments are reinforced laterally by the insertions of the Cardinal Ligaments, and anteriorly by the

Sub-Vesical Ligament which has attachments with the Vaginal fascia.

It is thus apparent that the upper Vagina relies on a stable Cervix for its support. The Cervix, in its turn relies on the Cardinal and Utero-Sacral Ligaments.

The postero-inferior portion of the Bladder rests on, and is attached by fascial attachments to the Sub-Vesical Ligament.

The Urethra also rests on the Sub-Vesical Ligament. It then passes through, and is firmly attached to, the Urogenital Diaphragm.

The Rectum, in the standing, or upright position lies postero-inferior to the Vagina. It may however bulge into the Vagina if the supports of the Posterior Vaginal Wall are weak.

Anatomy of Prolapse

The various types of prolapse can now be considered in relation to their deficient supports.

Prolapse of the Uterus occurs if the Cardinal and Utero-Sacral Ligaments elongate.

When the Uterus prolapses the angle of insertion of the ligaments into the Cervix becomes more acute. Two almost opposite forces are now acting on the Cervix. The weight of the Uterus is pressing downwards, and the ligaments are pulling upwards. Because of these two opposed forces the Cervix of a prolapsed Uterus tends to elongate.

When prolapse of the Cervix occurs, the anterior wall of the Upper Vagina and the Sub-Vesical ligament lose their upper support, become lax and bulge into the Vagina. The base of the Bladder and proximal part of the Urethra lose the support of the Sub-Vesical ligament and, in their turn, prolapse into the Vagina.

A Cystocoele can also occur without prolapse of the Cervix if the Sub-Vesical ligament loses its rigidity and becomes lax.

The proximal part of the Urethra will prolapse if the Sub-Vesical becomes lax. The lower part will not prolapse unless its attachments to the Ischio-pubic rami by the Urogenital Diaphragm are weakened.

The Posterior Vaginal Wall above the Levator Ani will lose its upper support if the Cervix prolapses. In the course of time two types of prolapse may occur.

1. High Rectocoele

The Rectum, finding no resistance anteriorly, bulges into the Vagina.

2. Enterocoele

The peritoneum in the anterior part or the Pouch of Douglas is firmly attached to the Supra Vaginal Cervix and the Posterior Fornix of the Vagina, in between the Utero-Sacral ligaments. The Pouch of Douglas is deepened by the pull of these attachments. Later, finding no resistance anteriorly, it bulges forward into the Posterior Fornix. As the Pouch of Douglas usually contains loops of Small Intestine, this type of prolapse is called an Enterocoele.

The factors which determine the type, or the relative degree of each type, which will occur are:

1. The strength of the posterior attachments of the Rectum, and the tone of the Rectal Wall.

2. The initial depth of the Pouch of Douglas between Rectum and Vagina.

A Low Rectocoele occurs when the Perineal Body is deficient. The lower part of the Rectum, having no anterior support, can bulge forwards into the Vagina.

Briefly the types of Genital Prolapse that occur are:

1. Prolapse of the Uterus.
2. Cystocoele.
3. Urethrocoele. The proximal or distal portions may prolapse singly or together.
4. Enterocoele.
5. High Rectocoele.
6. Low Rectocoele.

Causes of Genital Prolapse

Genital Prolapse is due to weakening of the supports mentioned above. This weakening can be produced by the localized trauma of Labour or by a generalized debility. Typically it occurs in three groups of women:

1. The young woman who has a series of Labours, followed by periods of excessive housework. The Labours tend to stretch the Cardinal, Uterosacral, and Subvesical ligaments and the upper Vagina. They may also produce tears in the Perineal Body and Urogenital diaphragm. Lack of rest following Labour prevents the pelvic supports from returning to their pre-pregnant state. The upright posture and the raised intra-abdominal pressure, which housework entails, tend to force the pelvic organs down into the Vagina.

2. At the Menopause the tone of the pelvic supports diminishes and prolapse tends to occur. This is probably due to Oestrogen withdrawal.

3. Post-Menopausal women develop Genital Prolapse because of the general loss of muscular tone and debility that tends to occur with advancing years. Often there is also a history, and evidence, of trauma produced during Labour many years previously.

Treatment

An Operation is indicated in the majority of cases.

A Pessary is indicated in those cases where an operation is not possible due to the Patient being a poor Surgical risk, or because she refuses an operation. A pessary is also advisable in the case of the young woman who expects to become pregnant in the near future.

General treatment to improve the general muscular tone and exercises to improve the Perineal muscular tone, are useful adjuvants to the more specific lines of treatment.

Pessary Treatment

Pessaries have been used ever since Genital Prolapse was first described.

Hippocrates used half a pomegranate (Littre 1851). Ambroise Parey used a wax covered ball (Ambroise Parey 1649). In more recent years the Ring, or Watch Spring, Pessary has become the pessary of choice. The Perineal Body must be present and the Levators not too widely separated in order that the Pessary will have a shelf on which to rest.

In cases where the Perineal Body is absent, it may be possible to perform a Perineorrhaphy. This is a short operation compared to the operative procedures which follow, can be performed under local anaesthesia, and will permit the use of a Ring Pessary later on.

If the Perineal Body is absent and a Perineorrhaphy is not possible, a Cup and Stem Pessary attached to a belt around the waist, can be used. A more recent type is the Gellhorn Pessary, This consists of a plastic cup and stem lying free in the Vagina (Gellhorn 1935).

Care of the patient wearing a pessary is necessary in order to prevent the occurrence of vaginal irritation, infection, ulceration or fistula formation. The important points are regular changing of the pessary, regular inspection of the Vagina by the Doctor, and regular vaginal douching by the patient.

Operative Treatment

Many operations have been devised for the treatment of Genital Prolapse. In order to be successful an operation must be anatomically sound. In the attempt to restore the pelvic anatomy to normal, the following procedures should be performed:

1. Shortening of the Cardinal Ligaments.
2. Amputation of the lengthened portion of Cervix.
3. Reconstruction of the bulging Anterior Vaginal Wall.
4. Reconstruction of the bulging Posterior Vaginal Wall and the torn Perineal Body.

There are two operations which include these procedures — the Manchester Repair and Vaginal Hysterectomy.

There are also a number of other operations which though not anatomically correct do cure certain groups of patients.

The Manchester Repair

In 1888 Donald, in Manchester, England (Donald 1908), and Olshausen and Schroder in Germany, began to perform an operation combining Anterior Colporrhaphy, Amputation of the Cervix and Posterior Colporrhaphy for Genital Prolapse. Previous to this date many operators, including Marion Sims (Marion Sims 1886), had been performing these procedures singly, but never all three together. Fothergill described the anatomical principles underlying the operation (Fothergill 1907). Sir Wm. Fletcher Shaw gave the operation its present name of Manchester Repair, and introduced it into the United States of America in 1933 (Shaw 1933).

During the operation an attempt is made to reconstitute the Subvesical Ligament by stitching the separated sheets of fascia together in the midline. The Uterus is supported by a stitch which passes through both Cardinal Ligaments and brings them together in front of the Supravaginal Cervix. The lengthened Cervix is shortened by amputating the Vaginal portion. The extent of the Posterior Colporrhaphy depends on whether the Rectocoele is High or Low. If an Enterocoele is present a portion of the Pouch of Douglas is excised, and the Peritoneum closed with a purse-string suture. The Uterosacral Ligaments can then be sutured in the midline to form a supporting shelf.

Shaw followed up 549 patients for a minimum period of three years. He found that the operation had been successful in 96% of cases. In women over 50 years of age, the Success rate was 97.6%. In women who had a Labour subsequent to the repair, there was no recurrence of the prolapse in 85%. His mortality rate was 0.23% (Shaw 1933). In a larger series consisting of 2152 repairs performed by a number of operators, the mortality rate was 0.37%.

Vaginal Hysterectomy

I have been unable to trace the originators of this operation. It was performed in Vienna by Schauta. A detailed anatomical description of the operation is given by Peham and Amreich (Peham and Amreich 1935).

The types of Vaginal Hysterectomy may be classified as follows:

1. Simple Vaginal Hysterectomy for disorders of the Uterus.

2. Vaginal Hysterectomy and Vaginal repair for disorders of the Uterus and Genital Prolapse.

3. Vaginal Hysterectomy and Vaginal Repair for uncomplicated Genital Prolapse.

The type of repair that accompanies a Vaginal Hysterectomy consists of:

1. Suturing the pedicles of the Fallopian Tubes, Uterine Arteries and Cardinal Ligaments in the midline.

2. Suturing the Uterosacral ligaments to the Cardinal ligaments.

3. An Anterior Colporrhaphy with reformation of the Subvesical ligament.

3. A Posterior Colporrhaphy.

4. Repair of an Enterocoele.

Many series of Vaginal Hysterectomies have been published. Averett, in a series of 2427 cases had a mortality rate of 0.24%. He also quotes Heaney's series of 1016 cases with a mortality rate

of 0.28%, and Kennedy's series of 2000 cases with a mortality rate of 0.02% (Averett 1945).

Series dealing with the results of the operation are less numerous due to the difficulty of following cases up.

Palmer in a series of 300 Vaginal Hysterectomies, had 4 recurrences of Prolapse. A failure rate of only 1.3% (Palmer 1948).

Danforth followed up 111 cases on whom he had operated. His failure rate was 18% (Danforth 1945).

Te Linde considers that Vaginal Hysterectomy, when used in the treatment of Genital Prolapse, gives an unsatisfactory result in 30% of cases (Te Linde 1948).

The Manchester Repair and Vaginal Hysterectomy Compared

The two operations have many points in common. The basic differences are that the Uterus is removed and the Cardinal ligaments sutured in the midline in a Vaginal Hysterectomy, whereas in a Manchester Repair only the vaginal portion of the Cervix is removed and the Cardinal ligaments are shortened by suturing a loop from either side in front of the Supravaginal Cervix.

Both operations have similar mortality rates.

The question to answer in deciding which operation to perform is, which provides the better support for the Vaginal Vault.

Statistics can be misleading. Anatomically both operations are sound. Mechanically the Manchester Repair has two advantages. The Supravaginal Cervix remains as a keystone to the Vaginal Vault. The two loops of Cardinal ligament, sutured in front of the Cervix, are a stronger support than two free ends of Cardinal ligament sutured in the midline.

It must be admitted however that a prolapse can recur following either operation. On this point it is generally agreed that it is easier to repair a prolapse following a previous Manchester Repair, than one following a Vaginal Hysterectomy.

In general it appears that a Vaginal Hysterectomy is indicated if there is Uterine Pathology associated with Genital Prolapse. Where prolapse exists alone, a Manchester Repair is the operation of choice.

Other Operations

Other operations which are performed for the treatment of Genital Prolapse are:

1. The Interposition Operation.
2. Richardson's Operation.
3. Le Fort's Operation.
4. Fixation Operations.

The Interposition Operation

This was originally performed by T. J. Watkins in 1898, and called the Watkins Transposition Operation. Shortly afterwards, Wertheim described a similar type of operation. It is now called the Interposition Operation:

Fundamentally it consists of bringing the Fundus Uteri down into the Vagina and suturing it into position below the bladder. The patient must be sterilized at the time of operation or be past the menopause. A pregnancy in such a uterus would be catastrophic.

It is used for the treatment of Cystocoele and Prolapse of the uterus.

Te Linde considers that this operation is less shocking and gives better results than Vaginal Hysterectomy. (Te Linde 1948). Brady followed up 115 patients on whom he had performed this operation. It was successful in 95.6% of cases. The mortality rate was 1.4% (Brady 1948).

This operation offends one's anatomical principles but may be of use where there is a large recurrent Cystocoele in a woman who has passed the menopause.

Richardson's Operation

This operation, described by E. H. Richardson, consists of: Anterior Colporrhaphy, Posterior Colporrhaphy, Amputation of the Cervix and Amputation of the Fundus Uteri. It is performed per Vaginam. The supravaginal Cervix is not removed. (Richardson 1937). I have been unable to find figures of the results of this operation.

Le Fort's Operation

This operation was originally performed by Neugebauer in 1867. It was later described by Le Fort in 1877 (Le Fort 1877).

It is really a partial Colpocleisis. The anterior and posterior Vaginal walls are partially denuded of mucosa and sutured together. A Perineorrhaphy is also performed.

It is used for Complete Genital Prolapse (Procidentia), in post-menopausal women with poor tissues, who might not survive a more lengthy operation.

Three series of Le Fort operations have been published since 1936:

1. 33 cases. There were two deaths due to Pulmonary Embolus. The operation was successful in 94.7% of cases. (Adair and Da Sef 1936).

2. 31 cases. No deaths. The operation was successful in 94.7% of cases. (Collins and Lock 1941).

3. 43 cases. No deaths. The operation was successful in 97% of cases. (Mazer and Israel 1948).

It appears that this operation can be successful in a large proportion of cases.

It has two disadvantages. The first is due to partial closure of the Vagina, which makes sexual intercourse impossible. The second is that if vaginal bleeding occurs, a provisional diagnosis of Uterine Carcinoma will have to be made and an Abdominal Hysterectomy performed.

Fixation Operations

In these operations the Fundus Uteri, or Round ligament, are stitched to the Recti muscles. They must always be combined with a plastic repair of the pelvic floor, as by themselves they do not cure genital prolapse. They are rarely used at present as they are unnecessary when a proper vaginal repair has been performed.

Conclusion

The ideal treatment for Genital prolapse consists of an operation which restores the pelvic anatomy to its normal, or pre-prolapsed, state. The Manchester Repair appears to come nearest to this ideal. The Pessary and several other operations, notably Vaginal Hysterectomy, do, however, have a place in the treatment of Genital Prolapse. The final decision, as to which treatment to use, can only be made when the Gynaecologist considers his patient, as an individual, with her own special tissues, and particular type of prolapse.

Summary

1. The Anatomy of the Pelvic supports is briefly described.

2. The changes in this Anatomy due to Genital Prolapse are described.

3. The common causes of Prolapse are mentioned.

4. The Treatment of Genital Prolapse by Pessary and by Operation is considered.

5. The conclusion is reached that the ideal treatment is by an operation which restores the pelvic anatomy to normal.

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Surgery

Case Report

Mitral Commissurotomy Preliminary to Removal of a Bronchogenic Cyst

James S. McGoey, M.D.
306 Medical Arts Building, Winnipeg

The surgical treatment of mitral stenosis by means of commissurotomy has given successful results, and a great number of patients have been symptomatically improved.[1, 2, 3] The reports from many different clinics are remarkably similar and have a high percentage of good results. Commissurotomy has definitely achieved an established place in the treatment of mitral stenosis. It is generally agreed that the best result will be obtained when the patient has pure mitral stenosis, which is characterized by prominence of the heart in the pulmonary conus area, an apical diastolic thrill, accentuation of the mitral first and pulmonary second sounds, the classical apical rumbling diastolic murmur and fluoroscopic or angiocardiographic evidence of mild to moderate left atrial and right ventricular enlargement.

The strikingly good results in properly selected cases have become almost legendary. Patients are being subjected to this operation more frequently than before, and also a wider range of patients is being considered for mitral commissurotomy. Thus, as we progress in the surgical treatment of this disease, more than just ideal cases will have to be considered for operation. Not only will patients with more advanced mitral stenosis and mitral insufficiency be evaluated, but decisions will have to be made concerning patients who in addition to mitral disease have another lesion requiring surgical interference.

The principle of the evaluation of the cardiac status of an individual and the medical management, improvement and stabilization of that patient prior to some surgical procedure is time honoured. Now that surgery is able to contribute to the treatment and improvement of the cardiac status by operative procedures, it seems logical that in the future, more patients will be seen in whom this type of cardiac management will be used in preparation for future unrelated surgical interference. Thus, some definite improvement is effected by commissurotomy, so that at a later date other definitive procedures can be planned. This same principle has been employed by many groups in performing mitral commissurotomy in the midterm of pregnancy. Cases have also been reported in which the mitral commissurotomy and associated lesion have been treated concomitantly.[4]

Occasionally the lesion associated with symptomatic mitral stenosis is intrathoracic. The case described below is that of a patient with mitral stenosis and a bronchogenic cyst. The latter was removed seven weeks after the performance of

a mitral commissurotomy, which improved his cardiac status for the second operation.

Report of Case

F. K., a 48 year old male steam-fitter, was admitted to hospital in October, 1953, complaining of marked dyspnoea of six weeks duration. He was well until 1942, when he was treated for pneumonia of the left lung. He was asymptomatic again, until 1945 when the pneumonia recurred and from then on he noted increasing exertional dyspnoea. He continued to work, however, until November, 1952, when the dyspnoea was moderately increased and he was forced to change to a less strenuous occupation.

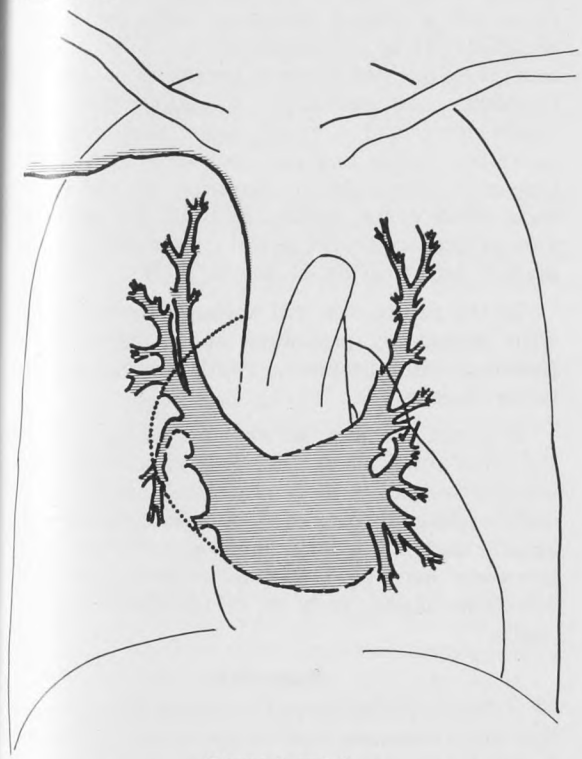


Figure 1 and 2

Angiocardiogram showing filling and enlargement of the left atrium and demonstrating the relationship of the right pulmonary veins to the right-sided mass. It pushes into the interval between the superior and inferior veins, partially obstructing the latter. Angiocardiograms taken at earlier intervals show no continuity of the mass with vascular structures.

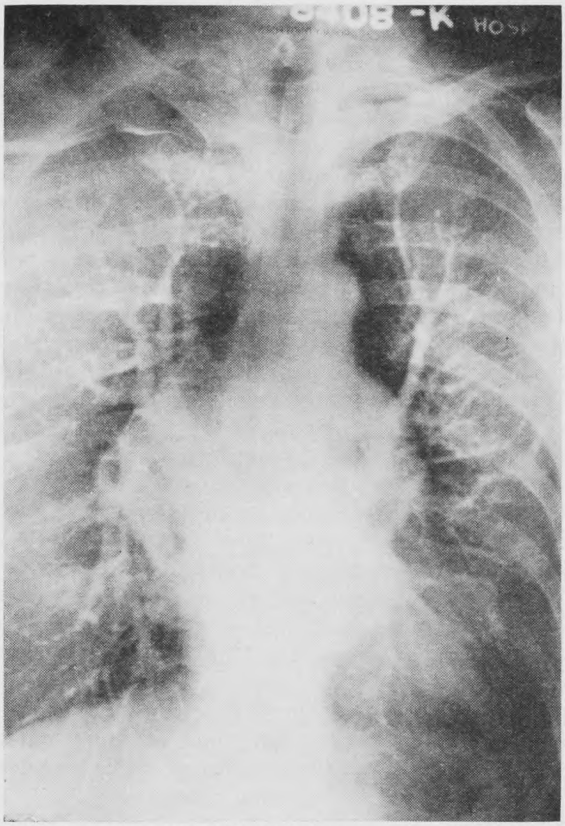
In September, 1953, he again consulted a physician because of dyspnoea, weakness, non-productive cough and orthopnoea. There was no hemoptysis or peripheral edema. An x-ray of the chest revealed a mass in the right hilar region, and he was admitted to Hospital in October, 1953.

Physical Examination

The patient was of asthenic habitus and became dyspnoeic on exertion. The pulse rate was 140 per minute and grossly irregular in rhythm. Blood pressure was 130/80 and respirations were 20 per minute. There was a palpable diastolic thrill over the mitral valve area, a diastolic apical murmur rough Grade 4, and a blowing systolic murmur Grade 2 and a loud pulmonic second sound. Blood chemical and hematologic studies were within normal limits and electrocardiograms revealed auricular fibrillation and right ventricular strain.

Fluoroscopy and Radiologic Findings

Fluoroscopy demonstrated a right hilar mass which was pulsatile and indistinguishable from the ascending aorta. The initial roentgenogram (Figure 1) revealed a sharply defined oval mass extending outward from the right cardiac border in the region of the right hilum. This was poorly defined in the lateral projection and appeared to overlie the right hilum. The cardiac



shadow was enlarged and there was a fullness of the pulmonary artery segment, causing a straightening of the left cardiac border. The lung fields were clear, but on the left side, there was thickening of the pleura in the costophrenic angle as well as flattening of the left hemidiaphragm. Angiocardiograms (Figure 2) demonstrated that the mass was distinct from the cardiac chambers and was probably a bronchogenic cyst or carcinoma.

Course in Hospital

The patient was maintained on daily digitoxin, twice weekly mercuhydrin and a salt-free diet, and on this regimen he showed improvement. Bronchoscopy revealed a lateral angulation of the right main stem bronchus to the right, but no endobronchial lesion.

Operations

Mitral Commissurotomy was performed on December 1, 1953 through the fourth left intercostal space with the patient in the antero-latero position. The pleural space was found to be obliterated, and a thickened fibrous peel, which enveloped the lower and much of the upper lobe was removed by decortication. The mitral valve area was explored digitally through the auricular appendage in the usual manner and was found to be stenosed to about 5 mm. in diameter. There was minimal regurgitation. The cusps were pliable and there was some calcification on the anterior leaflet. The valve was split by finger pressure to an opening of 25 mm. in diameter, without any increase in the regurgitation. Seven weeks later, on January 21, 1954, exploratory thoracotomy was performed through the right fifth intercostal space. An oval-shaped cystic mass measuring 8 x 5 x 5 cms. was identified in the posterior mediastinum behind the parietal pleura. It impinged upon the carina, which was markedly broadened and was closely adherent to the right main bronchus. Anteriorly and medially it was closely adherent to the wall of the left atrium. Laterally it bulged into the interval separating the superior and inferior pulmonary veins, partially obstructing the latter (See Figure 2. c.). Posteriorly it was in close apposition to the esophagus. It was separated by sharp dissection from the overlying structures and removed.

Pathologically the specimen was a thin-walled cyst filled with green, watery fluid and lined with ciliated columnar epithelium. There were also some attached anthracotic nodes.

The patient again had an uncomplicated post-operative course and was discharged from the hospital on February 13, 1954. When last seen on September 15, 1954 he was markedly improved and back at work. There was now no dyspnea, except on severe exertion, nor any orthopnea. There was no apical thrill or any diastolic murmur, but a Grade 1 mitral murmur persisted.

Discussion

This patient was admitted to the hospital because of an enlarged cardiac silhouette. Heart chamber studies by serial angio-cardiography demonstrated that the mass was distinct from the left atrium, superior vena cava, pulmonary artery and ascending aorta. It was then considered to be a mediastinal cyst or bronchogenic carcinoma, in either case demanding exploration. It was felt, however, that because of the severe disability associated with the mitral stenosis, he would not withstand a preliminary right thoracotomy for exploration of the mediastinal mass.

Mitral commissurotomy, and concomitantly, decortication of the left lower and left upper lobes were carried out. At a later date, it was possible because of his marked clinical improvement to remove a bronchogenic cyst which was in the posterior mediastinum, closely applied to the left atrium and partially obstructing the venous return of the right lung.

If this patient had not had such advanced mitral valvular disease, consideration could have been given to simultaneous surgical treatment of both lesions.[4] In the present day, with excellent anaesthesia, antibiotics and sound post-operative management, the desire of the patient and surgeon to escape a second operation must be carefully weighed. It is commendable to avoid a second operation, but not when it results in an increased morbidity and mortality. Certainly this patient would have had a much more dangerous post-operative course and may not have survived the extensive manipulation required to remove the right-sided cystic lesion, had he not been previously prepared with mitral commissurotomy and partial decortication of the left lung.

In the future, we will encounter more patients with pulmonary pathology and general surgical pathology and pregnancy with associated mitral valve disease.

It is not possible, as we have shown, to select the time intervals by clinical impression. However, as we gather more objective data, by cardiac catheterization, angiocardiography, electrocardiography and pulmonary function tests, physiologic processes may be better quantitated and better selections made, both as to time and extent of surgery.

Summary

1. The management of a patient with symptomatic mitral stenosis and a right-sided intrathoracic lesion is described.

2. Two operations were performed. First a left thoracotomy with decortication of the left lung and mitral commissurotomy and secondly removal of a ciliated bronchogenic cyst was carried out.

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"Problems of the Newborn Infant"

A series of case reports and commentaries from the files of the Winnipeg General, St. Boniface and Children's Hospitals, illustrating factors which affect the survival of the infant during his first week of life.

SERIES III

Infection in the Newborn*

A. J. De Pape, M.D., F.R.C.P.(C.)
Department of Pediatrics, University of Manitoba

Infection ranks as the fourth commonest cause of death in the newborn period and in Potter's review of close to 9,000 neonatal deaths it constitutes the major cause in approximately 13% of cases. It is very often missed until the autopsy table is reached, and this is so because of certain peculiar features inherent to this age group. These features involve the etiology, pathogenesis, immune response and signs and symptoms and make the diagnosis often quite difficult.

It is the purpose of this paper to review these features very briefly and to present a case of neonatal pneumonia.

Etiology

The etiology, except perhaps in the case of M. pyogenes var. aureus (Staph.) is likely to involve unusual types of organism and most frequently those belonging to what is often spoken of as the Colon group, namely Pseudomonas aeruginosa (pyocyaeneus, Aerobacter aerogenes, and Escherichia coli.

Pathogenesis

In addition to the usual portals of entry such as the nasopharynx and the skin the newborn is peculiar in having a moist umbilicus through which organisms can find their way into the body. It is this latter portal of entry which is often forgotten and which often leads to sepsis within the abdominal cavity or within the vascular system. This is the route followed by those pathogens which go on to cause the acute bacterial infections of the liver and gall bladder.

Immune Response

The immune response is different in the neonatal period from that of any other period in life. In essence it is the immunity of the mother so that, at best, the newborn baby can only be immune to those infections to which the mother is immune. The mother, in turn, transmits to the fetus only the antibodies of those infections to which she has actively acquired immunity, and this actively acquired immunity, by and large, concerns those pathogens in her immediate environment. This is an important point to remember when patients are transferred to Hospital because, while the mother may be immune to her own environment, she may have no immunity to pathogens prevalent in the hospital environment and therefore will be unable to transfer any immunity to this latter environment to her fetus. This latter is an argument which is often brought forth in favor of parturition at home instead of in the Hospital.

Another important feature is that the immunity which the newborn receives from the mother is strictly a "borrowed" immunity; it is humoral only and not cellular. By that is meant that, when the newborn acquires an infection for the first time against which he may have received antibodies from the mother, these antibodies which he has received from the mother are rapidly used up but the production of new antibodies by the baby's own body tissues is delayed because the infant's tissue cells have not yet been "trained" to recognize the infective agent as a pathogen and to form antibodies against it.

It is generally believed that the fetus does not produce any antibodies in utero, so that they are entirely passively acquired.

A final point is that, apparently, sometimes antitoxins only are received from the mother and no antibodies; this may be the case, for instance, in M. pyogenes infection in the newborn and could explain why some babies occasionally present with extensive pyodermas and still not show any evidence of being disturbed by their condition.

Signs and Symptoms

In the average case one does not get the same signs when an infection attacks a newborn as are seen in the adult. There is frequently no fever or the temperature may rise to 99 or 100 degrees. The W.B.C. count is normally high, and the differential, at least in the first days of life, shows a high polymorphonuclear preponderance in this age group. The heart rate is often used as a guide, and a tachycardia may sometimes be the only sign of infection; but if there is jaundice—a physiological process in the newborn—a bradycardia may sometimes be found instead. Many of the infections involve the respiratory system and sometimes a disturbance of respiratory rate or rhythm can be detected; this is not unusually a terminal event. Also there is often a disturbance of feeding habits with some emesis and anorexia, but, in their milder forms, these are a fairly common occurrence in relatively normal babies, particularly during the first week of life. In summary one has to be constantly thinking of infection because the signs are often not such as to suggest the diagnosis.

Pathology

By far the commonest lesions are found in the lungs and 80% of deaths due to infection involve

*This work is part of a neonatal mortality study project supported by a Dominion-Provincial Health Grant.

the respiratory system. Other systems involved are the G.I. tract (liver frequently), integument, C.N.S., G.U. tract, and others. It is well to remember that in many of the above the blood stream is also involved so that there is frequently a septicemia as well.

Following is the case summary of a newborn who died of pulmonary sepsis, but in whom other systems were involved as well.

Case No. 54-4743 — (Autopsy D.99)

Full term. Female. Birth Wt. 3,657 grams. The mother (Aet. 29 yrs.; Gr. 6; P.5.) had suffered from severe anaemia during the pregnancy, due, in part, to recurrent severe epistaxis and, also, to megaloblastic anaemia of pregnancy; she was admitted to St. Boniface Hospital two weeks before delivery and received during that period a total of 2,500 ccs. of blood by transfusion; this raised her hemoglobin from 5.4 gms. to 11.2 gms. on the day of delivery. On April 20th, 1954, she went into spontaneous labour and was delivered without difficulty, (L.O.A.). There was less than the usual blood loss associated with the delivery. The postpartum condition of the mother was good.

The baby breathed spontaneously within 30 seconds and was perfectly well until the 3rd day when she had a relatively short bout of moderate epistaxis, developed rapid respirations and became somewhat dusky at intervals. The liver was slightly enlarged and the spleen palpable. Hemoglobin 17.9; R.B.C. 5.6; C.I. 1.08; W.B.C. 14.6 with 44% polys and 39% lymphs A diagnosis of hemorrhagic disease of the newborn was made and Vitamin K administered. This episode lasted a few hours only and she then did well until the 6th day when she suddenly developed a fever of 101 degrees which rapidly increased to 106.4 degrees within 1½ hours. Respirations became rapid at 60-64 per minute and laboured, and there was cyanosis about the lips; a white tenacious discharge appeared at the nose.

Alcohol sponge baths brought the temperature down to 102 degrees within a half hour, and over the next 4 hours there was some improvement but the baby remained somewhat restless. On the 7th day and about 10 hours after the onset of the illness, respirations became gasping in nature at 84 per minute and there was marked subcostal indrawing particularly on the right. She was alternately listless and irritable, eyes were watering, the eyelids were red and edematous. She appeared anxious to feed but choked badly. A chest film revealed an ill defined shadow in the R.U.L. suggesting either partial collapse or consolidation but more likely the latter. Repeat W.B.C. remained unchanged. On the 8th day she appeared somewhat improved but still quite irritable when disturbed. A loose dark green stool was passed. Later on that day opisthotonus became noticeable but the C.S.F., apart from a total protein of 68 mgms. % was not unusual. On the 8th day condition deteriorated, respiration became rapid and shallow and marked cyanosis developed; the temperature rose from a previous average of 97 or 99 degrees to 100 and she expired.

Treatment had consisted of Streptomycin and Penicillin (SRD) ½ cc. on the 5th, 6th, 7th and 8th day of life; Terramycin 50 mgms. I.M. q.8.h. for the same period; Oxygen and parenteral fluids.

At autopsy the lungs were heavy and weighed 96 grams (normal, 54 grm.); they showed an extensive multifocal pneumonic process involving the parenchyma only, no lesions being seen in the laryngotracheobronchial tree. The infiltrate seen was essentially of a neutrophilic polymorphonuclear variety, filling many of the alveoli and infiltrating parts of the alveolar wall.

The other organs showed very early and mild infiltrations of the middle ears, leptomeninges and spleen, early fatty changes of the liver, and marked atrophy of the thymus. The adrenals showed a mild degree of tubular degeneration.

Cultures of the blood, right and left lungs, spleen, and C.S.F. were all negative, but the larynx grew a Pseudomonas aeruginosa and some hemolytic M.pyogenes.

In summary, then, this was a case of infection of the respiratory tract, probably associated with an initial bacteremia, but the organism could not be recovered at autopsy because of antibiotic therapy in vivo. Otitis media and a very early leptomeningitis was suspected as well.

Discussion

This case brings out some of the difficulties encountered in the diagnosis and management of infections of the newborn. It is believed likely that this baby already had an infection when she showed the first signs of illness, even though transient, on the third day and that had a diagnosis been made and therapy started at that time, the outcome might have been different. One is often loathe to subject a newborn to diagnostic procedures and antibiotic therapy on the basis of transient or relatively mild signs. **Nevertheless one should think of sepsis in all cases where signs are not clearly and completely explained otherwise and cultures of the throat, blood, C.S.F., urine or of any other visible septic lesion done as indicated.**

A final word should be said about treatment. The choice of antibiotics often poses a problem. Sometimes the pathogens cannot be isolated and sometimes one has not time to wait for culture identification and sensitivity studies. In such cases one should probably use the most innocuous form of therapy which will give the maximum of protection and a combination of Penicillin and Streptomycin I.M., at the present writing, is probably as good a choice as any. In those cases where

identification is possible one is guided by sensitivity studies. It is well to remember that one of the commoner pathogens in this age group, M. pyogenes, is notorious for being, not only extensively resistant to many and sometimes to all antibiotics, but also of an ever changing pattern of resistance as well. With respect to other pathogens it has been our most recent experience to find P. aeruginosa sensitive in vitro to Oxytetracycline and Tetracycline, A. aerogenes sensitive to Chloramphenicol and E. coli to Chloramphenicol and Oxytetracycline and Tetracycline.

Summary

Infections in the newborn have been reviewed briefly and in broad lines. Important differences occur in this age group in the etiology, pathogenesis, signs and symptoms and immune response. A case of sepsis involving mainly the respiratory system is presented.

The importance of early antibiotic therapy has been stressed and, where doubt exists about the presence of an infection, prophylactic antibiotic therapy suggested.

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Abstracts from the Literature

Hemorrhagic and Interstitial Pneumonitis with Nephritis. T. W. Parkin, I. E. Rusted, H. B. Burchell, J. E. Edwards. Am. J. Med., **18:** 220-236, 1955 (Feb.)

Hemoptysis was a prominent clinical feature in 7 patients believed to have had hypersensitivity states as evidenced by glomerulonephritis in all 7, and periarteritis nodosa in 4. In an occasional case, hemoptysis may be an expression of a generalized hypersensitivity state, and may so dominate the clinical picture as to suggest primary pulmonary disease.

The pathological findings are described in these 7 cases. Lesions were uniformly found in the lungs and kidneys. The immediate cause of death was considered to be asphyxia due to pulmonary hemorrhage. The lungs showed consolidation of large areas resulting from hemorrhage into the parenchyma. Four processes, common to all 7 cases, were acute necrotizing alveolitis with intra-alveolar hemorrhage, thickening of connective tissue in alveolar walls, the presence of prominent cuboidal cells lining many of the alveolar walls in areas of hemorrhage, and organization of blood in alveolar spaces. Other data suggests that the lesions may heal by resolution, leaving practically no recognizable residua; healed stages of the lesions may be seen in the absence of acute lesions; and that the pulmonary lesions are related to other allergic lung conditions. Uremia alone does not lead to these lesions.

Circumstantial evidence strongly favors the view that pulmonary alveolitis, with resultant pulmonary hemorrhages, and nephritis are considered to be manifestations of a hypersensitivity response.

A. G. Rogers.

Late Systemic Complications of Hydralazine (Apresoline) Therapy. J. C. Muller, C. L. Rast Jr., W. W. Pryor, E. S. Orgain. J.A.M.A. 157: 894-899, 1955 (March 12).

Hydralazine reactions occurred in 7 of 53 patients (13%) who received the drug from 4 to 23 months. Mostly the reactions appeared when the dose exceeds 400 mgm. daily for over 6 months. Recognition of the hydralazine reaction may be made clinically (fever, arthritis, pleurisy, pericarditis). Laboratory changes may be evident by depression of the bone marrow function, by the demonstration of L. E. Cells, by E.C.G. and urine abnormalities.

In 6 of the 7 patients, the hydralazine reaction resembled rheumatoid arthritis. It subsided with discontinuance of the drug. In one patient, L. E. cells developed, with fever, arthritis, pericarditis, pleurisy with effusion, resembling acute lupus erythematosus. This subsided after treatment with cortisone and corticotropin. L. E. cells were observed in 2 patients with the hydralazine reaction, and in 2 patients who were asymptomatic. Reported reactions to hydralazine include fever, pancytopenia, acute psychoses, gastro-intestinal bleeding, and a collagen-like illness, resembling rheumatoid arthritis when mild, and acute systemic lupus erythematosus when severe.

Hydralazine (Apresoline) should be reserved for patients with severe hypertension states, when the morbidity and mortality overshadow the dangers of the drug. The use of Apresoline alone is only partially effective. Apresoline and hexamethonium constitute one of the most effective forms of therapy for hypertension.

A. G. Rogers.

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Radiology

Cholografin

A. J. Glazebrook, M.D.,* A. C. Abbott, M.D.** and R. Hastings-James, M.D.***

Cholografin is an intravenous medium for cholangiography which was first introduced into Germany about three years ago. We have used it in more than 60 cases, and the purpose of this paper is to illustrate both its value and its limitations by describing the clinical features of some cases in which results of interest were obtained.

Cholografin contains iodine, but the iodine is firmly bound to the molecule, and it seems unlikely that iodine sensitivity needs to be taken into consideration in using the compound. After intravenous injection it is excreted by the liver cells and the concentration in the bile rapidly rises to a degree sufficiently great to give satisfactory opacification of the larger biliary passages, the test resembling the intravenous pyelography examination. This high concentration in the bile is independent of the gall bladder which visualizes at a much later stage if it is present. About ten per cent of the material is excreted through the kidneys, but if, for any reason, the route through the liver is hindered this percentage may increase sufficiently to be visible as a pyelogram.

In a recent paper, (1) we suggested that the manufacturer's instructions regarding the timing of the X-ray films and the rate of intravenous injection be modified. The first opacification of the bile ducts may take place almost immediately after injection, and to wait 10 minutes before taking the first film, as recommended, may result in the substance being seen in the small intestine, instead of in the biliary tree, so rapid may be the flow. On the other hand, a concentration great enough for visualization may be delaysd for 60 minutes. We have given the intravenous injection in three minutes instead of ten, finding this better, both from the point of view of the patient and the quality of the pictures obtained. We have found the skin test for sensitivity unhelpful. As an index of liver function the excretion of cholografin does not seem to parallel the usual laboratory tests. Further details as to the methods we employ will be found in the above mentioned paper.

Plate 1 shows the appearances in a normal subject. Opacification of the major hepatic and the common bile ducts appears first in ten or twenty minutes. In about sixty minutes the gall bladder is seen, its shadow becoming denser in the next hour or so. A fatty meal may be used to test gall bladder contractility. The shadows are rarely as opaque as those produced by Telepaque in Graham's technique.

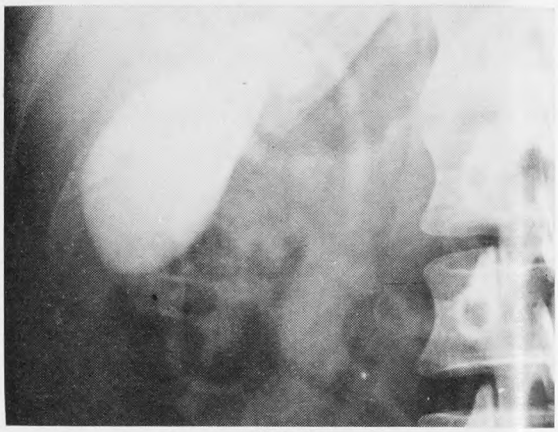

Plate 1
Cholografin visualisation of normal gall bladder and bile ducts.

In the following presentation we try to show some of the ways in which cholografin has been of assistance to us, illustrative cases being mentioned in support of the statements made. The most interesting results are summarised in Table I.

In the first two cases, the intravenous test is considered negative. Only post-operative cholangiography reveals the hidden stones. It can be seen that the test is by no means infallible.

Case 1: A 55 year old man came into hospital complaining of right upper quadrant pain coming on 15 minutes after meals and lasting for 15 minutes, of about four months duration. The pain was sharp and steady in nature, with no radiation, and at times it was sufficiently severe to make him roll about. He noticed it about twice a week;it never occurred before meals or at night time. Fatty foods exacerbated it, and for three weeks before admission he had avoided them and had been free of pain. He was too easily satisfied by his meals, feeling distended soon after eating. There had been no constipation, no jaundice, and no change in stool or urine color. He had lost some 13 lbs. in weight during the past year, but he attributed this to his poor appetite.

After admission his icteric index was found normal, but he had excessive urobilin in his urine. A Graham's test failed to visualise the gall bladder. On cholografin examination the common bile duct was estimated to be of normal width, although at the upper limit. No calculi were observed.

At operation chronic cholecystitis was seen and the gall bladder removed. No calculi were observed either in the gall bladder or common bile

*Consultant in Gastro-Enterology, St. Boniface Hospital, and Associate Professor of Physiology and Medical Research, University of Manitoba.
**Surgeon, St. Boniface Hospital, and Assistant Professor of Surgery, University of Manitoba.
***Radiologist, St. Boniface Hospital, and Assistant Professor of Radiology, University of Manitoba.

Table 1 — Summary of the most interesting results

Case	Cholecystectomy	Graham's Test	Cholografin	Final Diagnosis	Value of Cholografin Test
1) Man Aged 55	.....	Non-visualising gall bladder	Normal common bile duct	Chronic cholecystitis and stone in common bile duct	Unhelpful. Only post-operative cholangiogram revealed hidden stone
2) Woman Aged 28	.....	Non-functioning gall bladder with calculi	Normal common bile duct	Cholelithiasis. Stones found in dilated common bile duct	Unhelpful. A dilated common bile duct with calculi did not show
3) Man Aged 37	1949	.....	Patulous sphincter of Oddi	Gastric ulcer	Excluded dyskinesia of Sphincter of Oddi
4) Woman Aged 52	1951	.....	Dilated common bile duct (Plate 2)	Dyskinesia of Sphincter of Oddi	Demonstrated a dilated common bile duct
5) Man Aged 84	.....	Non-visualising gall bladder	Dilated common bile duct; probable stone	Pancreatitis	Gave evidence of an associated lesion; a dilated common bile duct
6) Man Aged 71	.....	Non-visualising gall bladder	Dilated common bile duct; probable obstruction	Pancreatitis	As for case 5
7) Woman Aged 45	.....	.....	Normal common bile duct; calculus right kidney	Normal common bile duct; stone in right kidney	Nephrolithiasis diagnosed by pyelogram effect; bile duct disease excluded
8) Woman Aged 65	1939	.....	Dilated common bile duct; probable stone	Hiatus hernia and bile duct stone	Dilated bile duct shown
9) Woman Aged 47	1954	.....	Common bile duct stone	Common bile duct stones	Demonstrated common bile duct stone but missed stone in the ampulla of Vater shown by post-operative cholangiogram
10) Woman Aged 49	1951	.....	Dilated common bile duct with 3 stones (Plate 3)	Dilated common bile duct with many stones	Showed dilated common bile duct containing stones
11) Woman Aged 65	1953	.....	Dilated common bile duct with stones	Cardiac failure and common bile duct stone	As for Case 10
12) Woman Aged 70	.....	Non-visualising gall bladder	Normal common bile duct	Stone in a dilated common bile duct	Indicated bile duct disease but failed to reveal the calculus found at operation

duct, the latter being probed, but it was found impossible to pass a probe or a catheter through the ampulla. T-tube drainage was instituted, and two post-operative cholangiograms with 35% diodrast demonstrated the presence of a calculus approximately 1 cm. in diameter just proximal to the ampulla of Vater.

Non-visualization of the gall bladder, when using telepaque, is nearly always indicative of disease. In this case the Graham test was the more helpful. The value of cholangiography can be plainly seen from this experience and we have had built a special cassette-tunnel, so that films can be wound into position during the operation and exposures made, after filling the common bile duct with contrast material.

Case 2: A 28 year old woman was admitted to hospital during the fourth month of her pregnancy. She was exceedingly anxious to keep the baby, but she gave a history of recurrent attacks of typical gall bladder colic, which had doubled her up and had radiated to between the shoulder blades, over the last 2 years. She had also suffered from 3 episodes of a severe pain, situated deeply in the epigastrium, which she described as being like an afterbirth pain, and which had been associated with very high serum amylase values. These three attacks had been accepted as being due to pancreatitis.

A Graham's test before admission had shown a non-functioning gall bladder containing calculi. A cholografin test was done chiefly to check the width of the common bile duct before operation, for record and follow up purposes. This gave a poor visualization of the duct, but as far as it could be seen it was not abnormal in any way. Liver function tests showed hepatic impairment and in view of all the findings, laparotomy was advised.

At operation the gall bladder, full of small stones, was seen and removed, but in addition to this the common bile ducts were found to be thickened and grossly dilated and contained calculi, one being discovered in the ampulla of Vater.

Our next two examples show the value of the value of the investigation as applied to the post-cholecystectomy syndrome. In case 3 we find a demonstrably non-functioning sphincter of Oddi 5 years after operation, which contrasts with the usually accepted dictum that the sphincter regains its tone within six months of cholecystectomy.

Case 3: A man aged 37 was admitted to hospital with a long history of stool frequency dating back to an attack of dysentery while he was working in a Russian Prisoner of War camp under starvation conditions in 1940. In 1949 a laparotomy was carried out because of a complaint of persistent abdominal pain and the gall bladder was found to be densely adherent to both the liver and the duodenum. It was removed after division of the adhesions. There were no calculi and no pathological report on the condition of the gall bladder which was described by the surgeon as being large, thin walled, and greenish in color.

After the operation, he continued to have right upper quadrant pain, varying in intensity, reaching its maximum at noon, radiating through to the back, and not related to food, posture, or bowel movement. Examination of the stools in another hospital had shown a steatorrhoea, but after admission, repeated fat estimations on a fixed intake failed to confirm this. His blood findings were normal. There was no faecal occult blood. He continued to have a rather loose bowel movement after every meal, without mucous or pathogens.

A "post-cholecystectomy" syndrome was suspected, perhaps related to dyskinesia of the sphincter of Oddi, but cholografin studies showed so rapid a flow of the medium into the small bowel, that visualisation of the common bile duct was not obtained, as at that time we were following the instructions of the manufacturers and taking the first film at ten minutes after the end of the intravenous injection. Morphine gr. ¼ given parenterally had no effect upon the ability of the sphincter to retain the dye. A view of the common bile duct was only obtained on the third examination when films were taken immediately after the intravenous injection.

The examination therefore revealed a patulous sphincter of Oddi to be present 5 years after cholecystectomy, the sphincter being unaffected by morphine gr. ¼. Dyskinesia was excluded as a cause of his symptoms. Whether the dribbling of bile from a patulous sphincter could have played any part in his lienteric diarrhoea is a matter for speculation.

In case 4 a distended common bile duct is the sole finding; this is confirmed at operation and a diagnosis of dyskinesia is made. (Plate 2).

Case 4: This unfortunate woman had her gall bladder removed in 1951 for gall bladder stones; her common bile duct was reported at that time as being normal.

Ever since the operation she had suffered from abdominal pain especially after fatty foods, fried foods, sour foods and fresh bread; together with flatulent distensions and belching. The pain had a rather diffuse location across the front of the epigastric region, it was also referred through to her back.

An intravenous cholangiogram showed a greatly dilated common bile duct, with a considerable quantity of dye in the radicals of the hepatic ducts. There was no filling defect suggestive of a stone. The cholografin did not pass readily into the duodenum and appeared to be held up by the sphincter of Oddi.

At operation the common bile duct was found to be as large as a man's thumb, but no stones were present. The duodenum was opened and the sphincter of Oddi dilated. The sphincter was extremely small and very tight and it was therefore laid open for ½ inch over a grooved probe, and the mucosa of the common duct sutured to the mucosa of the posterior duodenal wall. She made an excellent recovery and so far has been free of symptoms.

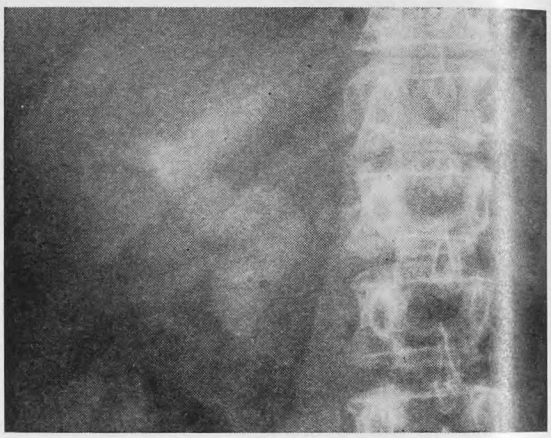

Plate 2
Dyskinesia of Sphincter of Oddi.
Common bile duct dilated.
No stones.

In both cases 5 and 6, acute pancreatitis having been diagnosed on clinical grounds and the blood amylase findings, a dilated common bile duct, with possibly some obstruction, is shown with the cholografin test. Intravenous cholangiography may prove a valuable method of investigating pancreatitis when the acute symptoms have passed.

Case 5: An 84 year old man was admitted to hospital with acute right upper quadrant pain radiating through to the back. He gave a history of similar attacks which had responded to medical treatment occurring at intervals during the preceding three years. On examination there was tenderness and rigidity in the right upper quadrant but no mass was felt. His serum amylase was 2742 Somoygi units, (normal 60-160 units). He was treated medically and his serum amylase fell to 800 units and finally to 177 units; at the same time his abdominal pain and tenderness lessened and then disappeared. When he was symptom-free, a Graham test was done and this failed to visualise

the gall bladder. Cholografin examination, however, showed a dilated common bile duct with a probable stone at its lower end.

Case 6: This 71 year old man had been troubled by attacks of very severe pain lying deeply in the epigastrium and radiating to the left upper quadrant for six weeks before his admission to hospital. It had also been referred to the back. The pain had been more or less constant, but food definitely made it worse, and he got relief by lying down in bed. It was colicky in character. Injections of morphine had been given for its relief.

Pancreatitis was suspected, and on admission his serum amylase was 800 Somoygi units. With bed rest and restricted diet his pain lessened and disappeared, and his serum amylase level fell to 200 Somoygi units (normal 60-160). When he was

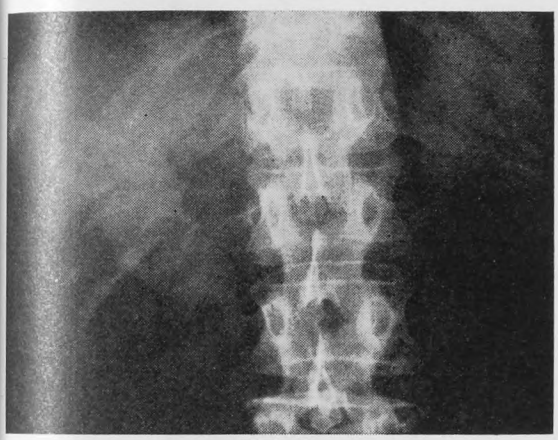

Plate 3
Dilated common bile duct containing stones.

better a Graham's test failed to visualise his gall bladder, but a second test using a double dose of Telepaque revealed a poor concentration of dye with a delay in emptying.

A cholografin examination, however, showed a dilated common bile duct and the slow build-up of concentration within it suggested some obstruction.

In case 7 intravenous cholangiography establishes the position of a stone in the right kidney by its pyelogram effect, as well as showing the common bile duct; the presence of the stone being confirmed at operation and the common bile duct being found normal.

Case 7: A 45 year old woman had a cholografin examination done together with other investigations thought necessary to find the cause of her epigastric pain referred through to the back.

The intravenous cholangiogram showed that a stone was present in the right kidney owing to the pyelogram effect; a good visualization of the common bile duct was also obtained and it was seen to be draining well. At laparotomy the common duct was found to be normal; a stone was removed from the right kidney.

Case 10 illustrates the value of cholografin; common bile duct stones are proved present 4 years after cholecystectomy.

Case 10: A middle aged woman, who had her gall bladder removed in 1951, came to Hospital complaining of repeated attacks of biliary colic of 3 months duration.

Intravenous cholangiography showed a dilated common bile duct measuring 1" in diameter. Within it, 3 stones were seen (Plate 3). At operation, 3 large stones and many small stones were removed from the dilated common duct. A post operative cholangiogram with diodrast through a T-tube drainage 7 days after laparotomy revealed a common bile duct still dilated; no stones were observed within it.

Summary

We have found intravenous cholangiography with cholografin of use in 10 of over 60 cases examined. It must be regarded an ancillary to the ordinary Graham's test; and the chief indications for its employment lie in the investigation of the post-cholecystectomy syndrome, or as an additional examination when Graham's test fails to visualise the gall bladder. It may be useful in discovering the cause of pancreatitis, when the acute symptoms have abated.

The test is not infallible, and the demonstration of an apparently normal common bile duct with cholografin does not exclude biliary pathology.

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Children's Hospital, Winnipeg

Ward Rounds

Edited by Wallace Grant, M.D.

Internes' Seminar — Children's Hospital

October 8, 1954

"Heart Failure in Childhood"

(Continued)

Part II

Discussion:

(a) Pathological Physiology — Dr. J. B. Armstrong

Dr. Lauer in his fine exposition has stolen most of my thunder. I don't know whether he remembers it or not, but when he was in third year Medicine, I gave him a lecture on this subject. It was strangely similar to what he has outlined in his slides and presentation, although his is much more complete than mine was. If you refer to the Oxford dictionary, you will find that congestion mean "excessive accumulation" and failure means "inability to meet the demands", just like on a final examination. Thus congestive failure of the heart presumably means excessive accumulation of something because the heart is failing to do its job. How does it get that way? Dr. Lauer has already pointed out that the demands may be increased in any of a number of ways, such as by coarctation. The prime work of the heart is to raise blood to the pressure of the arteries, that is the aorta or the pulmonary arteries, and the actual propelling of this blood constitutes, at best, somewhat less than a tenth of the work of the heart. In severe muscular exercise, I am sure, this may become closer to a third. In other words, much of the work of the heart depends on the pressure in the great vessels, and conditions which increase the pressure, in the pulmonary circuit or the aorta, will put a great demand on the heart. There are some other factors that may be mentioned. For instance, many children with congenital heart disease are polycythemic and this means greater viscosity of the blood, greater resistance and hence more work for the heart. Similarly, if the children are anemic the heart will have to work harder because cardiac output is increased. The oxygen-carrying capacity of the blood being decreased, more blood has to be supplied per unit of time to the periphery when there is anaemia. Dr. Lauer has drawn our attention already to the other large group of conditions. Thus the demand may not be increased, but the sufficiency of the heart may be decreased by rhythm disturbances, affections of the epicardium (such as pericardial effusions) affections of the myocardium (and he listed these in great detail) and affections of the endocardium, including the

valves, and endocardial shunts. These are all conditions under which the heart is not going to work as well or as efficiently as normal, and therefore it is less likely to meet the demands placed upon it.

If the heart fails to meet the demands placed upon it, one of the first things that appears to happen is that the elimination of salt and water is not carried out as effectively as in the normal. There is a good deal of argument as to how this comes about. I don't intend to, or pretend to throw any new light on the problem. I think it was Dr. Merrill at Emory University about 1944 who found that in adults the amount of sodium excreted by a person in failure was much less than by a normal individual or the same individual when taken out of failure. He found that their glomerular filtration rate was not appreciably decreased, and that their renal blood flow may or may not be decreased, (in more severe failure it usually is decreased) There is then, a mechanism by which the renal tubules reabsorb a higher percentage of the sodium filtered than is normally the case. It has been postulated that this may be due to adrenal cortical factors, but such have not been substantiated by many workers in the last three or four years. It has not been shown, for instance, that there are increased 11-oxysteroids in the urine in people going into congestive failure, although we know perfectly well that if we give something like DOCA, sodium is retained. It has also been suggested that there is some retention of water because the posterior pituitary substance (anti-diuretic hormone) is increased. However, Dr. Perry and his associates (in fairly recent experiments in the Department of Medical Research of this University) were not able to show that there was any significant increase in the serum A.D.H. in people in congestive failure although such an increase in excretion in the urine has been described by other workers. In short then, the evidence for a hormonal mechanism to account for the reabsorption of sodium is not very convincing. In spite of this there are those who favour it.

With the accumulation of sodium, in order to keep the body fluids roughly isotonic there is an associated accumulation of water. The water and sodium retained increase blood volume, and this is, at least, one of the reasons why the venous pressure increases and presumably why the cardiac silhouette, as seen by X-ray, increases. Furthermore, increase in venous pressure can affect renal excretion of sodium, as has been shown by a good many people. Bradley was one of the first — by putting cuffs around the abdomen, and increasing the intra-abdominal pressure, he found that the renal excretion of sodium was decreased. Similarly Freedman in Vancouver, working with dogs, has ligated veins at various levels throughout the body and has found that you get an increased sodium retention even if you ligate the femoral veins. So there is more to this factor of increase in venous pressure than merely increase in the pressure of the renal veins. In fact, Tinsley Harrison, in discussing unpublished work in 1949, stated that he had at that time people in his department who were putting cuffs around the neck, and by increasing the pressure in these cuffs and increasing the venous pressure in the head, he found that sodium excretion was markedly decreased. It is obvious then that it is an extremely complex mechanism that controls the renal tubules. I have discussed this matter in this way because Stead for instance, had patients on a limited salt intake who were on the verge of congestive failure, and when he increased their salt intake in a very modest way (about 5 grams a day), they went into congestive failure. Their weight gain could be graphed quite dramatically, and yet their increase in venous pressure, measured several times a day, came considerably later. In other words, although venous pressure can certainly have a secondary effect in congestive failure, I think the primary effect is renal. However I do not know what may be the mechanism for the increased reabsorption of sodium, when the heart begins to do less than what is expected of it.

(b) Pharmacological Therapy of Heart Failure —

Dr. Mark Nickerson (Professor of Pharmacology and Medical Research).

It has been suggested by Dr. Israels that I tell you how to handle the problem of congestive failure in children now that it has been defined. However, the classification of heart failure which has been presented indicates that there are perhaps twenty different problems, of which we are capable of handling effectively only two or three. It is much easier to talk about the pharmacology and the therapeutic aspects of congestive heart failure in the adult because the condition is much more uniform and in addition, more work has been done on adult patients. Perhaps I should start out with a brief consideration of what Dr. Israels referred to as the pharmacology of the failing heart. This, for practical purposes, is the pharmacology of digitalis. In this discussion I will use the term digitalis to include all the commonly employed cardiac glycosides.

As has already been mentioned, the major factor in the development of congestive heart failure is an inadequacy of the ejection capacity (contractile capacity) of the myocardium of the ventricles. This can be due to an inherent inadequacy of the ventricular muscle, or to an increased demand upon it due to some factor such as hypertension (as in coarctation of the aorta in children). In either case it is a relative insufficiency. It is proper to focus our attention almost entirely upon the contractile capacity of the ventricles in congestive heart failure. Factors such as heart rate or diastolic filling may be involved occasionally, but they are only rarely of major importance. In spite of the many cycles of philisophy relating to digitalis action, I think we now have come back to the point of view that its primary effect, and the only one of clinical significance in the average case, is its effect on contractility. Certainly digitalis decreases the size of the dilated heart, but this is not necessarily a part of its beneficial action. The increase in contractility has in the past been attributed to a return of the myocardial fibres to an appropriate length (you may recall the diagram of Starling's law in your physiology test books), an "increase in diastolic tone". Evidence is now very adequate, particularly since the work of Cattell and Gold[1] on papillary muscles, that the increase in contractile strength induced by digitalis can be quite independent of changes in the resting length of the muscle.

Changes in cardiac rate are also of only secondary importance in the response to digitalis, except in some cases of arrhythmia. In cases of auricular fibrillation the effect of digitalis on the conduction system of the heart is important in slowing the rate, but in the majority of cases of congestive heart failure the increased vagal activity which acts to slow the rate is secondary to the development of compensation. An abnormally high rate without decompensation is rarely slowed by digitalis in subtoxic doses. Slowing of the rate **per se** is not of major importance in the development of compensation. In one study of children with active rheumatic heart disease, Schwartz and Levy[2] were able to produce a consistent reduction in rate by digitalization, but this did not improve the congestive failure in a single patient. Except in the case of the arrhythmias, a decrease in heart rate is a measure of improvement only when it is a result of improved myocardial contraction.

The action of digitalis in congestive heart failure thus appears to be very simple, and it is up to a point. Digitalis increases the contractile force

of the myocardium, its capacity to do work. But how does digitalis improve the capacity of the myocardium to do the work? Here we have very little information. A logical possibility to investigate would be the ability of the myocardium to utilize various substrates in the production of energy. This initially looked like a promising line of attack, because we do know that the failed myocardium puts out less work per unit of oxygen consumed than does normal heart muscle. When we go beyond that point and look at the substrates utilized, at the production and storage of high energy phosphate bonds, etc., we find that the failed myocardium behaves in exactly the same manner as the normal, and the administration of digitalis does not alter this pattern.

There are two approaches which have recently given us possible indications of the mechanism of action of digitalis. However, it will be many years before we know whether either of these is a correct evaluation of its action. One, relates to the potassium balance of the myocardial fibres. We have known for a long time that when the activity of the myocardium is stopped and then restarted, the first contractions are very weak and they gradually increase in strength until a plateau at or near maximal contraction capacity is reached. Hajdu,[3] working with Szent-Gyorgyi's group, has noted that during each contraction there is a loss of potassium from the muscle fibres and that during the diastolic interval before the next contraction only a part of the released potassium reenters the fibres. In other words, the gradient of potassium concentration across the cell membranes gradually changes as the strength of the contraction increases. A point is finally reached where the gradient remains more or less constant, and that point is associated with maximal contractility of the myocardial fibres. He also observed that, to some extent in the normal, but particularly in the failed heart, digitalis slows the re-entry of potassium into the fibres and tends to cause the gradient to reach a steady state more rapidly and to be more stable. This effect on the passage of potassium across the cell membrane may or may not be the action of digitalis which is of importance in congestive heart failure, and I am presenting it here merely as one of the more intriguing possibilities.

Another possible mechanism of action of digitalis has been suggested by the work of Dr. Robb at Syracuse.[4] Actomyosin, the active contractile protein complex in heart as well as skeletal muscle, can be formed into threads either by extruding it into a medium in which it is insoluble, or by making a monolayer on a liquid surface and then rolling it up to form a thread. Many of the studies which can be done on isolated strips of heart muscle can also be done on these actomyosin threads. Of course one has to be much more careful with the threads. Instead of working against grams of weight, the threads are required to lift only milligrams or fractions of milligrams. However, the threads have many of the same characteristics as heart muscle; they contract when ATP is added and the work produced by this contraction is directly related to resting length as is the work output of the myocardium. Dr. Robb found that in the case of the actomyosin from heart muscle, but only to a limited extent with the actomyosin from skeletal muscle, the addition of digitalis to the fluid surrounding the fibre increases the amount of work it can do. The improved performance is particularly prominent when digitalis is applied to threads which previously have been "failed" by a barbiturate or by some other procedure. This is a quite different concept from that of Hajdu, and it suggests that the digitalis is acting directly on the contractile protein of the myocardium. These data also are presented merely as items of interesting information without any commitment as to whether or not this is the ultimate explanation of the activity of digitalis in congestive heart failure.

Now I should like to say a bit about some of the more practical aspects of the pharmacology of the failing heart. This is much more thoroughly understood in the case of adult heart failure than it is in that of children, partly because of the much higher incidence of the former and partly because it is much more homogeneous than heart failure in children. There are, however, a number of observations on adults which can be applied to the treatment of children. We know that in adults atherosclerotic and hypertensive heart disease respond best to digitalization. At the other extreme, cor pulmonale and heart failure associated with acute infections (diphtheria, etc.) respond poorly. There are two features of the cases which respond well which are worth noting. They are conditions in which the heart is chronically overworked, and which involve primarily the left ventricle, although the right side of the heart may be subsequently involved. These two factors must be considered in any analysis of the use of digitalis in congestive heart failure in children.

Among the causes of heart failure in children, congenital heart disease is usually listed first. Heart failure due to morphological defects usually responds poorly to digitalis, except for congestive failure associated with coarctation which usually responds well. In most types of congenital heart disease we are dealing with a primary insufficiency or overwork of the right ventricle. In coarctation of the aorta it is the left ventricle which is primarily involved, and this difference may explain the more favorable response to digitalis in cases of coarctation. However, the fact that cases of congenital heart disease respond poorly to digitalis

on the average does not mean that this drug should not be tried. Occasionally, for reasons that are not clearly understood, the response is very good.

If subendocardial fibroelastosis is considered as a primary myocardial disease, it is clear that it should be differentiated therapeutically from other congenital heart disease. This condition tends to respond well to digitalis. I can not give you a definite basis for this difference in response, but it may be that the residual normal muscles fibres of the left ventricle are chronically overworked. Nedas and his co-workers in Boston[5] have recently reported a series of about ten cases of subendocardial fibroelastosis, well over 50% of whom responded very favorably to adequate doses of digitalis.

Another important cause of congestive failure in children, in many areas, is rheumatic fever. I must say that I was most surprised to note that so few cases of rheumatic fever were included in the very nice outline of your cases which Dr. Lauer presented. You are most fortunate that rheumatic fever is rare in this area. During the rheumatic fever season in Utah, where I previously worked, as many as 50% of our pediatric beds were sometimes occupied by children with rheumatic fever, and it is a most difficult and discouraging condition to treat. The response of congestive failure in rheumatic heart disease to digitalis is highly variable, and there are almost as many different opinions of this type of therapy as there are authors writing on the subject. This divergence is probably due to the varying amounts of acute myocardial, or pancardial involvement, and of residual organic damage from previous rheumatic attacks. In the adult one not infrequently sees congestive failure in a rheumatic heart which is almost entirely due to old organic damage. These cases respond fairly well to digitalis, although not as well on the average as do those cases of failure due to atherosclerosis or hypertension. It is probable that the poorer results in children are due to the fact that failure due to rheumatic fever in children almost always involves a large component of acute rheumatic involvement. My own impression is that the greater the factor of acute myocardial involvement in a case of rheumatic failure, the less likely it is to respond favorably to digitalis.

In addition to the difficulty in predicting the effect of optimum digitalization in heart failure in children, it is much more difficult to evaluate dosage and the adequacy of therapy than is the case in adults. The dosage range in children is much wider than it is in adults. Nadas[5] has found that children who respond favorably to digitalis may do so at a digitalizing dose of anywhere between 0.01 and 0.05 mg. per pound of body weight. This is not only a range of 500%, but also suggests a considerably greater requirement for digitalis in the child. However, this difference is not nearly so impressive if one evaluates dosage on the basis of body surface area, usually a more reliable indicator of drug requirements than is weight, when one is dealing with organisms considerably different in size. In adults the average digitalizing dose of digitoxin administered over a period of 36 hours (moderately rapid digitalization) is about 0.75 mg. per square meter. In children it is usually between 0.5 and 1.25 mg. per square meter. Nadas found that most, but not all children who were going to respond well to digitalis did so at a dosage below 0.03 mg. per pound in patients less than two years old and at a dosage below 0.02 mg. per pound in older children.

In children the effective dose is usually closer to the toxic dose than it is in adults. As a matter of fact, those children who respond well usually also show some evidence of toxicity. It has been noted frequently that patients responding to the lower dosages also tend to develop signs of toxicity at lower dosages than do other patients. This parallelism between the development of therapeutic and toxic effects is perhaps related to the extent to which different hearts accumulate digitalis from the body fluids. The concentration of digitalis in heart muscle is always greater than that in the circulation or in most other body organs, and this accumulation is probably related to the rhythmic activity of the heart. Skeletal muscle also may be induced to accumulate digitalis by rhythmic stimulation.

The fact that there is little margin between the therapeutic and toxic doses of digitalis in children does not mean that the dose should be pushed until the child is prostrated with vomiting and diarrhea. However, it is usually desirable to persist until electrocardiographic signs of overdigitalization appear. Below the toxic level the signs of digitalization are very similar in adults and children; depression of the S-T segments, depression or inversion of the T wave, and in particular, shortening of the Q-T interval as a fraction of cycle length. Any one of these three signs may appear first, but they do not necessarily mean adequate digitalization in children. Any one of the above signs may appear at 50 to 75% of what is ultimately determined to be the optimum dose. The electrocardiographic signs of digitalis toxicity are rather different in children than in adults, ominous ventricular rhythm and extensive prolongation of the P-R interval are less common, whereas conduction defects such as bundle branch block are much more frequently observed.

In summary, congestive heart failure in children presents a much more difficult therapeutic problem than it does in adults. The situation is usually

more acute and more dangerous to the patient's life. Each case is a law unto itself, being modified by the type of heart disease involved and by individual factors which have not been adequately analyzed. Certain types can be expected to respond better to therapy than others, but the factor of individual variation is sufficiently important to warrant a trial with digitalis even in types of heart disease which respond poorly on the average. Finally, adequate digitalization of children is complicated by the rather wide range of dosages required in individual cases and by the fact that in children adequate digitalization is very close to or in the toxic range.

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(c) **Dr. H. Medovy**: I would like to congratulate Dr. Lauer on a fine, painstaking practical presentation. He has shown us how important the problem of heart failure in infancy really is. The diagnostic possibilities lead us into areas not touched on by internists and cardiologists who see only the adult patient. In the infant we must consider congenital heart disease, paroxysmal tachycardia, and "primary myocardial disease" (a term which includes subendothelial fibro-elastosis), Glycogen storage disease of the heart, aberrant left coronary vessel, and idiopathic myocarditis. In the older child, congenital heart disease and rheumatic carditis constitute the problem. Diagnosis in infants may be very difficult. The presence of marked respiratory distress may focus attention on the lungs instead of the heart.

Diagnosis was made by the pathologist in about 30% of our cases in infants. This is of course not good enough. With improved diagnostic tools and with the increased incentive of an active, cardiac surgical centre in the hospital, we should do much better in the future. During the next year we hope, with the assistance of government grants, to carry out an intensive study of a series of children with undiagnosed cardiac states in the public schools.

Editorial

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

Failure in Medicine

Failure in Medicine has many connotations. To the undergraduate about to write his finals or to the graduate preparing for higher examinations, it sounds an ominous note. To a practicing physician, whose waiting-room is not overflowing with patients, and whose income is below the reported (statistics of the Income Tax Department) average, it connotes the very opposite to professional and financial success. To the philosophically minded it implies the frustrating failure to attain the ultimate ends of Medicine, that of cure and prevention of disease.

In order to allay the anxiety of the reader, who may be depressed by these dismal topics, it may be stated, that the latter are not the subject of this editorial comment, and shall not be referred to again. The failures under consideration are not those of people, but of organs. The former were mentioned only in order to emphasize the anthropomorphic character of the term "failure", which appears to endow anatomical structures with qualities of goal seeking, usually ascribed to volitional, purposeful acts of human beings. This, of course, is not the first time that we encounter in Medicine expressions and terms that smack of anthropomorphism. The "educated" finger of the gynaecologist is an old timer. "Splenic aggressiveness" is a recent one introduced by R. M. Bird to designate hypersplenism. It may be now only a matter of time before thyroid arrogance, adrenal timidity, bone marrow backwardness, liver laziness and pituitary snobbishness find their way into medical writing.

Interestingly, the antithesis of the failure of an organ, namely its success is practically never referred to in medical literature. The body economy, unlike that of society, does not encourage excessive zeal and ambition. A highly "successful" hyperfunctioning organ is as bad as one who falls down on the job.

Failure, as applied to an individual organ, (the heart), was first used by the Alexandrian physician, Herophilus (4th century B.C.). Since then for many centuries the heart was the only privileged organ officially permitted to fail. No other failure was recognized. When doctors spoke of failure they did not need to qualify or supplement the term. It was taken for granted that the heart was the culprit. It is only within recent memory that failure of lungs or kidneys, liver, adrenals, pituitary, gonads, bone marrow, and brain has been recognized and accorded full respect, if not equal status with that of the heart.

The term "failure" is by no means self-explanatory. Indeed, it is not even a clearly defined one. Some use it interchangeably with insufficiency to indicate inadequate performance of function. Others reserve it for the late and final stages of insufficiency, when as a consequence of the bankruptcy of the organ all the compensatory mechanisms fail to function, and the whole homeostatic machinery of the organism breaks down. Thence the synonymous — decompensation.

The flexibility of the term "failure", as applied to organs, is well illustrated in the paper "Manifestations of Liver Failure", by Dr. J. H. Martin (published in this issue). In this article Dr. Martin discusses the pathophysiology and the clinical picture associated with the breakdown of the metabolic functions of the liver, which he designates as "liver failure". Since the chief functions of the liver are those concerned with metabolism, it is impossible to quarrel with this use of the term. Yet, one would do well to remember that the liver is also the organ of biliary excretion, as well as the site of three complex circulatory systems. Failure of excretion of bile due to intrahepatic biliary obstruction may lead to jaundice and cholemia before parenchymal changes become manifest. Failure of the hepatic circulation due to portal cirrhosis will often result in ascites long before metabolic liver functions are grossly deranged. Thus we may have three types of liver failure occurring independently, concurrently, or in succession, namely the metabolic, the biliary, and the circulatory each posing its own problems of diagnosis and treatment. The latter, of course, takes the matter out of the realm of pedantic quibbling into that of practical medicine. The subtleties of diagnosis are, thus, not without pragmatic implications, for while metabolic and excretory failure can be treated only with medical palliation, the circulatory lends itself to surgical procedures, e.g. porto-caval anastomosis, with the view of creating a shunt, and thus reducing the inflow through the portal tract, or magnesium trisilicate application (advocated by J. L. Madden)[1] for the promotion of adhesions between the liver and the diaphragm with the view of increasing hepatic outflow.

Objections similar to those in the case of the liver, could also be raised against the use of the term failure in relation to other organs, which share with the liver the characteristic of multiplicity of functions. The lungs, for example, perform the tasks of ventilation, oxygenation of blood and transfer of gases. They can fail in either one or all of these functions. Similarly, the pituitary

with its multiple hormones may present a variety of forms of decompensation. The bone marrow may display an aplastic anemia, a leucopenia, a thrombocytopenia or a pancytopenia — partial or total failure.

Having gone along thus far, and, perhaps, conceded reluctantly the inadequacy of this contentious term when applied to organs with more than one function, the perplexed reader will, no doubt, turn with confidence to the old standby—the heart. Surely, the faithful pump with its singleness of purpose and long tradition of decompensation cannot fail to support the validity of the concept of "failure". The heart has many parts and many properties, but they all subserve one function—the ejection of blood. Ability or inability to discharge this function should, then, be a simple clear-cut matter of observation and analysis, leaving no room for futile hair-splitting and semantic exercises. Yet, here it is—the doubt, and its weapon, the sharp knife of analysis getting down to the matter of the heart.

What is heart failure? Dr. J. B. Armstrong in his comments (published in this issue of the Review) defines it as "inability to meet the demands". This inability of the heart to meet the demands of the tissues for oxygen—carrying blood, is manifested clinically by well known symptoms and signs. The clinician recognizes them without difficulty. He also observes and differentiates two major variants—left heart failure and right heart failure, which he does not regard as a dichotomy, but rather as a subdivision or sequence of events. He may also notice that while most of his decompensated cases have cold hands and feet and respond well to digitalis, some have warm extremities, and derive little, if any, benefit from the glycosides. Somewhat puzzled, he turns to the physiologist, who assures him that here again he is not dealing with two separate entities, but two variants—low and high output failures, the latter, as opposed to the former being characterized by increased circulatory rate, decreased arterio-venous oxygen difference and increased output. The fact that the latter negates the very basis of the "failure" concept, namely, inadequacy of the cardiac output, does not seem to worry the physiologist, for, as he rightly explains, inadequacy is a relative term and although the output may be high in absolute terms, it is too low to satisfy the increased demand. Nor is the physiologist at a loss to explain the low renal blood flow, responsible for sodium retention and congestive phenomena, in the face of a high output. The explanation lies in the "shunting of blood away from the kidney due to a disproportionate loss of resistance in the extrarenal portion of the systemic vascular bed"[1].

Despite these reassurances, the clinician often feels uneasy about the whole thing. Nor are all physiologists serenely happy about it. Some are gracefully bowing out. Youmans[2] in his review of the subject substitutes the word "circulatory" for "heart" in describing the high output syndrome. He regards the latter as primarily peripheral, a result of decreased peripheral resistance, whether it be due to arterio-venous fistula or generalized vasodilatation.

Another source of perplexity in the study of heart failure is constrictive pericarditis. This condition is manifested by congestive phenomena, which clinically resemble right heart failure. Yet, in view of the purely obstructive nature of the disease and the absence of intrinsic myocardial pathology, it is not usually regarded as a form of cardiac decompensation. The logic of this argument, however, is by no means irrefutable, for if one accepts the forward failure theory with its definition of heart failure as a metabolic state due to salt retention, then one must consider constrictive pericarditis a legitimate contender, since salt retention has been shown by some observers to be a factor in this condition.

It becomes thus apparent that failure of a unifunctional organ like the heart is fraught with as many difficulties of definition and comprehension as that of a multifunctional organ like the liver, for the difficulties are basic. They are inherent in the mismated marriage of a functional concept of failure to a structural one of an organ. They could probably be lessened, if not obviated, if we were to think in terms of failure of function, e.g. ventilation, excretion, oxygenation, cerebration, rather than structure.

Moral: Not all marriages are made in Heaven, or, failure is no success. Ed.

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1. Madden, J. L.: Surg. Gynaec. Obstet., 1954, 99: 385.
2. W. B. Youmans: Mechanism of High Output Circulatory Failure. Annals of Int. Med., 414: 749 (Oct., 1954)

Manitoba's Medical Men

XVI. Government in Sickness Insurance

The Executive of the Manitoba Medical Association discussed at their last meeting a new trend in governmental thinking about prepaid medical plans. The Government of Alberta plans to subsidize medical care by entering into an agreement with the Medical Services (Alberta) Incorporated and other similar organizations in the province.

The amendment to the Hospital Treatment and Services Act reads as follows:

"The Minister may enter into an agreement with Medical Services (Alberta) Incorporated, or any insurance organization, company or society to pay a subsidy in an amount not to exceed one-third of the cost of providing for the residents of Alberta a policy or scheme of insurance covering medical, surgical and obstetrical services, on the condition that the cost of such policy or scheme of insurance to the purchaser is reduced in proportion to the amount of the subsidy."

The Council of the College of Physicians and Surgeons, after a discussion with the Minister, agreed with the proposals for the introduction of the plan, and as a result of this, the plan may become effective July 1, 1955.

On the basis of the plan, the Government will contribute $7.00 per person subscribing to the M.S.I. which will be about ⅓ of the premium. The other ⅔ will be paid by the subscriber, and, as a result, the premium will be reduced by this amount. No exclusions for age or pre-existing conditions are inherent in the plan, but a waiting period will be required for certain conditions.

One of the objections to the plan is that there is no provision made for those unable to pay the ⅔ premium required. This would include the indigents and those in the lower income groups.

A most important fact emerges from this proposal and that is that the Government of Alberta is willing to participate in a voluntary sickness insurance plan. Another point emerges, and this is of considerable importance, namely, that the Alberta Division of the Canadian Medical Association is not entering into this agreement, but the College of Physicians and Surgeons.

Finally, the question of extra billings may not be satisfactory to the Government and the profession may not wish to relinquish this right.

Another problem which does not concern the doctors of Alberta is the effect this subsidy will have on insurance companies or societies which are in the field.

Here in this province the problem was introduced in the legislature. A bill to amend the Health Services Act reads as follows:

"The Act is amended by adding thereto, immediately after section 32 thereof, the following section:

32A (1) Any municipal corporation, in lieu of erecting the whole or part of the municipality into a medical care district or establishing a district jointly with another municipality, may,

(a) as provided in The Municipal Act, enroll all, or any group, of the residents of the municipality as subscribers of Manitoba Medical Service;

(b) by by-law authorize the making of an agreement between the municipal corporation and Manitoba Medical Service; and

(c) make such an agreement, in such form and containing such conditions as the council may approve, whereby Manitoba Medical Service will undertake to arrange for the furnishing by duly qualified medical practitioners of surgical, obstetrical, and medical care and service, or any one or more of those kinds of care and service, and including or excluding medicine, for the residents of the municipality so enrolled.

(2) Where an agreement is made with Manitoba Medical Service, as provided in this section, whereby the residents of the whole municipality, or of a designated part thereof, are enrolled as subscribers of Manitoba Medical Service, the municipality or the designated part thereof shall be deemed to be included in a medical care district for the purpose of section 33."

And, again, another bill reads as follows:

"The Act is amended by adding to Division III of Part IV thereof, immediately after section 455 thereof, the following section:

455A. A municipal corporation, by by-law, may

(a) enroll all or any group of the residents of the municipal corporation or any part thereof, as members in Manitoba Hospital Service Association or Manitoba Medical Service; and

(b) provide for the payment of the fees for enrollment by all such residents or by any group of residents, or levy a rate therefor either against all the assessable property in the municipal corporation or against the assessable property of any group of residents who are enrolled, or partly against all the assessable property in the municipal corporation and partly against the assessable property of any group of residents who are enrolled, as the council of the municipal corporation in its discretion may determine; and for any of the above purposes may enter into an agreement with Manitoba Hospital Service Association or Manitoba Medical Service, or both of them."

At the end of March 1955 this legislation became legal, so that now municipalities will be permitted to levy taxes to cover the cost of prepaid medical care. This would be of great importance in covering medical care of indigents.

A further amendment to the last bill mentioned may include commercial carriers.

The possible implications of this new legislation on the Manitoba Medical Service, are difficult to assess, but it is safe to say that the closest scrutiny will have to be made of all legislation concerning Manitoba Medical Service which covers neither the indigents or the very low income groups and in addition to that has a means test for the high income groups. Those in the latter group may object to the use of public funds to support a plan of sickness insurance for which they are not eligible.

A full report of all the implications involved in these governmental subsidies to medical care will, no doubt, be forthcoming from the Board of Trustees of the Manitoba Medical Service.

L. A. Sigurdson, M.D.

In Appreciation

Sir Alexander Fleming
M.B., B.S., F.R.C.P., F.R.C.S., F.R.S.

On March 11th, 1955, the career of a famous bacteriologist, Sir Alexander Fleming, ended with his death at the age of 73. Fleming's discovery in 1928 of penicillin, the first antibiotic used for the successful treatment and cure of certain bacterial infections, changed the practice of medicine as much as any other discovery in medical history.

Fleming, the son of a Scots farmer, was born in 1881 in Lockfield in the Ayrshire village of Darvel and his early life was apparently uneventful. An elder brother, who was a physician, probably influenced Fleming's choice of a career, when he decided to study medicine at St. Mary's in London; it has been suspected, however, that the presence of a championship football team rather than the academic prestige of that famous institution was responsible for Fleming's choice of a school. Fleming had a pleasant personality. He was modest and unassuming, with a quiet voice and manner; he enjoyed the companionship of his fellows, yet he was stubborn and had the courage to uphold his convictions.

Following his graduation Fleming accepted an appointment to the faculty of St. Mary's Hospital as a teacher in bacteriology under the direction of Sir Almoth Wright, but when war with Germany was declared shortly afterwards, he joined the R.A.M.C. as Captain. He was stationed at the 13th General Hospital at Boulogne and during this period studied the effect of different antiseptic agents in the treatment of wounds. He finally reported that, while some antiseptics were effective as germicidal agents, unfortunately the majority of them also produced marked tissue destruction.

At the end of the war Fleming returned to St. Mary's Hospital as Bacteriologist, where, in addition to the routine duties of his post he continued his investigation for a less toxic wound disinfectant. It was during the course of this study that he discovered a germicidal enzyme in tears, lyzozyme, that would destroy a few types of bacteria, but which, unfortunately possessed little or no germicidal effect on more pathogenic organisms.

In September 1928 while examining a plate culture of staphylococci Fleming made his now famous observation of the inhibition of the growth of staphylococci in the area surrounding a colony of a contaminating mold, a penicillium. The natural reaction of a bacteriologist under such circumstances would be to discard quickly and to sterilize the contaminated plate in order to avoid contamination with the mold of succeeding plate cultures. Fleming, however, much interested in the zone of bacterial inhibition and also in the apparent bactericidal effect of the penicillium upon staphylococci decided to investigate the phenomenon further; later he also demonstrated that the broth in which a pure culture of the penicillium was grown contained a substance that inhibited bacterial growth, even in exceedingly high dilutions. He named this substance Penicillin.

In 1929 Fleming published the results of his experiments in the British Journal of Experimental Pathology, but the medical prefession showed little interest in the discovery. Fleming nevertheless continued his investigations and illustrated by the inoculation of animals with penicillin the absence of toxic effects in animals; the same lack of toxicity was demonstrated in human beings following treatment of superficial wound infections with penicillin.

The lack of interest of the medical profession in Fleming's work with penicillin was probably the result of the concurrent development of the sulfonamide drugs, which having proved their bacteriostatic efficiency against a number of bacteria were then considered the most important medical discovery of the time. The search for and the development of other similar chemotherapeutic drugs overshadowed Fleming's discovery of penicillin.

The onset of the Second World War stimulated interest in the treatment of wounds and an Oxford Research team of scientists was appointed to check Fleming's work with penicillin. Howard Florey and Ernst Chain, two members of the team, proved Fleming's claim that penicillin was a highly efficient bactericidal agent and mass production and the use of the antibiotics soon followed their

report. A direct result of Fleming's discovery of the antibiotic effect of penicillin was the stimulation of the attention of other scientists to that particular field and the discovery of many other antibiotics produced by different molds.

Although Sir Alexander Fleming during the past few years received many tributes for his famous discovery, probably the greatest was the honour conferred by an appreciative Sovereign when he was Knighted in 1944.

J. C. Wilt, M.D.

Coronach

When asked by the editor to write a requiem for Dr. Hunter, based on our long friendship, I began as never before to try to analyze our mutual attraction.

To me there was a similarity between him and another Scot whom I liked and admired for his qualities: Dr. Thornton, who practised in a small country town, and made the mistake, to my mind, of going into politics. Both men were cultured, well read, with only one standard of conduct.

Dr. Hunter shunned the spotlight and would not accept positions involving leadership in policies or organizations, yet it was remarkable how rapidly his qualities won him the respect of those individuals and groups to whom he had formerly been but a name.

He did not write a great deal, but in speaking you recognized that he was master of his subject. His reading was mostly done in the early hours of the morning for he was one of those who could do with less sleep than you or I can. In summer, he was up soon after dawn to work in the garden, which he loved more for its beauty than for the rarity of its flowers. Music gave him much happiness and his liking for it seems to have developed in Germany where one could attend grand opera at little cost. Outside of medical publications, his reading consisted mostly of history and biography; his familiarity with the speeches and writings of the great statesmen of the Empire during the last two centuries was the fruit of those studies.

He was one of a large family but seems to have lost touch with them since coming to Canada. Life in Aberdeen University followed the usual custom of medical students there: hard work on a diet of oatmeal and herrings. If he had any bursaries or scholarships to help him out, he has not told me, but then there is probably much that he has not. He once told me that I was the most incurious man that he had known, which no doubt cemented our friendship. What he wished me to know, he told me, assured that I would not ask questions about matters which were his own affair.

He was neither an atheist nor agnostic but belonged to no church, and at one time freely discussed the tenets of various religions. He always entered on discussions with an open mind, carried them on without heat, and never lost his temper. He was gifted with a sense of humour which enriched many of his tales, including those of his misfortunes.

He was somewhat critical of certain trends in modern medicine, not that he was opposed to change, but that pharmaceutical houses introduced new discoveries to credulous physicians before there had been opportunity for a full test of their values, and that some operations were performed for the glorification of the surgeon rather than for the benefit of the patient. He used to quote cases which shocked him, where operations were performed on dying people, when he would have tried to smooth the passing. He admired the surgeon who rated judgment above technical skill.

It was interesting to note the thoroughness with which he made an initial examination or acted as consultant. I used to threaten my old friend Archbishop Matheson with a visit from Dr. Hunter. His Grace would have summed up the Doctor as "too blooming wholesale". He did not expect his complaints to be taken that seriously.

He shared with me the pleasure of walking, and if time was no object never used a car. He had an electric closed car in the early days of the motor car and when he discarded that, never replaced it with the gasoline type.

My farewell has not been in the fashion of such an article, but it would be unfair to my friend to make use of well worn platitudes. He was always forthright in thought and action and would recognize the weakness almost amounting to insincerity. I miss him and my fellow members will feel with me that he has done much by his example to support the code of ethics which we strive to maintain.

E. S. Moorhead.

Obituaries

W. G. Beaton

To have known Grant Beaton was indeed a privilege. The community and his professional colleagues have suffered a profound loss in his departure. He has left behind him a lasting monument to his memory in the hearts of hundreds of people.

That most precious of human attributes, which we call kindness, belonged to Grant in very full measure. Integrity of the highest order was an integral part of him. These characteristics were never paraded, but were rather concealed under a thin mask of brusquerie; but no mask could conceal the delightful twinkle in an eye glinting with an almost everpresent sense of humor.

There was no compartment in his mind allotted to dissembling. His words were forthright, few and to the point. This characteristic of precision, caused him to strip his professional opinions completely bare of any unnecessary scientific verbiage. His investigating mind kept him fully apace with the march of medical science; and he was quick to embrace all new ideas, interpreting them in terms applicable to his patients, with the result that he inspired confidence in those seeking his aid.

To the younger medical men, he lavishly gave his time and service in assisting them.

A skilled Surgeon, and excellent Internist, a wise counsellor and a valued friend, has left our ranks to receive the accolade at the hands of the Great Physician Himself. We loved him greatly and we shall miss him profoundly.

Athol R. Gordon, M.D.

Dr. Charles Hunter

One of the most scholarly of Winnipeg physicians, Emeritus Professor of Medicine Dr. Charles Hunter died on March 18, aged 82. He came to Winnipeg in 1904 with a reputation of learning and diagnostic acumen and went on to win further distinctions. For nearly fifty years he was an outstanding consultant in internal medicine. He was one of the early members of the Winnipeg Clinical Society which after a stimulating career merged with an older and more conservative organization in 1921 to form the present Winnipeg Medical Society.

During the first World War he served overseas from 1915 to 1919. By 1916 he had his majority and was a member of the Medical Board at Folkestone and a little later was made a lieutenant colonel.

Returning to Winnipeg he was in 1927 appointed Professor of Medicine in the University of Manitoba, but administrative detail did not appeal to him and he resigned after a year yet retained his position on the honorary attending staff of the Winnipeg General Hospital. He had an extensive consulting practice and his opinions in difficult and obscure cases was sought by practitioners throughout the Canadian west. In 1930 he was honored by being made a Fellow of the Royal College of Physicians of London.

Born in Scotland he was educated in Scotland, England and Germany, but never lost the Aberdonian quality. He continued in active practice until 1952. He is survived by his widow.

R. B. M.

Central District Medical Society

On February 23, 1955, a "One Day Rural Refresher Course" was held in Portage General Hospital.

Chairman for the day was the president of the Medical Staff, Dr. G. C. Fairfield.

Doctors present included: Dr. G. M. Black, Dr. J. J. Y. Ch'uai, Dr. G. C. Fairfield, Dr. G. H. Hamlin, Dr. J. W. Kettlewell, Dr. M. E. F. Koziol, Dr. G. H. Lowther, Dr. T. W. D. Miller, Dr. G. T. McNeill, Dr. R. E Renaud, Dr. J. C. Rennie, Dr. G. V. Sutton, Dr. C. M. Thomas, as well as the visiting clinicians: Drs. J. N. Briggs, C. C. Ferguson and J. M. Kilgour.

Clinical cases were presented for discussion and comment. Cases presented included gout, renal colic, common duct obstruction, Reynaud's disease, arteriosclerotic gangrene, varicose ulcers, diaphragmatic hernia, mitral stenosis, congenital heart disease and acute tracheolaryngo bronchitis. The comments and discussions were most interesting and instructive.

Dr. Kilgour later delivered a paper on current views on antibiotic drugs and Dr. Briggs a paper on acute tracheolaryngo bronchitis. Both were ably presented and extremely thought provoking. Dr. Ferguson, who throughout the day had upheld the surgeon's reputation, presented some of the aspects and problems in the surgical care of heart disease. Particular emphasis was placed in the selection of cases that might be suitable for surgery.

During the day, the Portage General Hospital was host to the group at luncheon. For dinner and liquid refreshments we adjourned to the Portage Hotel about 5.30 p.m.

Dr. Macfarland brought greetings from the Provincial Association and related some of the recent problems that our executive body is coping with.

G. C. Fairfield, M.D.,
President.
C. M. Thomas, M.D.,
Secretary-Treasurer.

College of Physicians and Surgeons of Manitoba

Council Meeting (Cont.)

October 16, 1954.

B. Communication from the Registrar, C.P. & S., British Columbia.

The Registrar presented a communication from the Registrar of the College of Physicians and Surgeons of British Columbia enclosing an immigration pamphlet from the Department of Citizenship and Immigration, outlining requirements of the various provinces for registration. This pamphlet is issued by immigration officers overseas to interested physicians, and since many of the provinces have changed their regulations since the pamphlet was issued, corrections were requested. Before an alien physician is granted a visa to come to Canada to engage in his profession, he must present evidence that he has been in contact with one of the provincial registrars in Canada. Dr. Macfarland suggested that it might be wise to withhold information concerning the requirements for registration until the applicant arrives in Canada.

The Registrar was given permission to provide the Registrar of the College of Physicians and Surgeons of British Columbia with the information requested, to be co-ordinated with information received from other provinces, in order that a new pamphlet may be prepared for distribution in Europe.

C. Communication from the Executive Secretary of the Michigan State Board of Registration in Medicine.

The Registrar presented the following resolution for information, which was received from the Executive Secretary of the Michigan State Board of Registration in Medicine:

WHEREAS, the actions of the National Board of Medical Examiners of the United States have been and are incompatible and in violation of Act No. 237, P.A. of 1899, and Acts amendatory thereto, the Medical Practice Laws of Michigan,

BE IT RESOLVED, that on and after November 1, 1954, the Michigan State Board of Registration in Medicine will not accept or endorse the Certificate of Examination of the National Board of Medical Examiners of the United States as a basis for registration and licensure to practice medicine in Michigan.

D. Communication from the Chief, Division of Narcotic Control, Department of National Health and Welfare.

For information, the Registrar presented the following communication in part which was received from the Division of Narcotic Control, Department of National Health and Welfare:

"As you are no doubt aware, some amendments to the Opium and Narcotic Drug Act were made at the last Session of Parliament and since that time new Regulations as provided for by the Act have been made by Order-in-Council and have become effective September 15, 1954.

"One of the main items in the new Regulations, is the definition of an 'oral prescription narcotic product.' In brief, it is medication containing a narcotic drug in combination with two or more non-narcotic medical substances in recognized therapeutic dose and not in a form intended for parenteral administration. This is the type of medication a pharmacist may dispense on the strength of an oral prescription issued by a physician, dentist or veterinary surgeon, providing, of course, certain procedures are followed. Sales of all straight drugs as well as narcotic preparations not coming within this category may only be sold by a pharmacist upon receiving a signed and dated prescription. Moreover, prescriptions calling for any narcotic medication, whether written or orally given, cannot be repeated by a pharmacist."

E. Communication from the Honorary Secretary of the Canadian Medical Association, Quebec Division.

A communication was received from the Honorary Secretary of the Canadian Medical Association, Quebec Division, inquiring concerning the number of practising physicians in Manitoba and regulations of this province concerning alien physicians. Council agreed that the Registrar forward the required information.

8. Inquiries.

None.

9. Notice of Motion:

"THAT Part VII, Section 4B of the By-laws, Rules and Regulations be amended by deleting $5.00 from the third line and replacing it by $10.00."

10. Motions of which notice has been given at a previous meeting.

None.

11. Unfinished Business.

None.

12. Miscellaneous and New Business.

A. Payment of Janitor.

(Refer Item 4D, Report of the Finance Committee.)

B. Amount to be Paid to Council Members for This Meeting.

(Covered by Part VII 4A of the By-laws, Rules and Regulations. No motion required.)

C. Motions re Salaries and Amount to be Paid to the Manitoba Medical Association Each Month.

(Salaries of office assistants covered in Item 4D, Report of the Finance Committee.)

Motion: "THAT the Registrar's salary be Three Hundred Dollars ($300.00) per month, and the Treasurer's salary be Six Hundred Dollars ($600.00) per annum." Carried.

The Registrar advised that the rental of the combined business office had been increased and would probably mean an increase in the monthly payment to the Manitoba Medical Association from the C.P. & S.

Motion: "THAT the amount of Eighty Dollars ($80.00) per month be paid to the Manitoba Medical Association for services, and that it be referred to the Liaison Committee for further consideration." Carried.

D. Adjournment:

1:30 p.m.

Motion: "THAT the meeting be adjourned." Carried.

Registration Committee

October 27, 1954.

Personal Interviews

Christopher Francis Wolkenstein, M.B., B.S., U. Melbourne, 1947.

John Silinsky, M.D., l'Aurore U., 1948.

Dieter Kirchheim, M.D., Johann Wolfgang Goethe University at Frankfurt-am-Main, 1951.

William Harold Ho Asjoe, M.D., Creighton U., 1953.

Alexander Johannes Bozyk, M.D., U. Vienna, 1944.

The following were unable to remain to be interviewed by the Committee, but had met with the Registrar on various occasions:

Kai Harold Pihl, M.D., College of Medical Evangelists, 1946.

Jazeps Teodors Beldavs, M.D., U. Latvia, 1940.

Enabling Certificate Deferred

Wasyl (William) Shahariw, Certificate (in place of diploma), Donetz Medical College, 1941.

Enabling Certificate Granted

Donald Dave Robert Ballyk, M.D., St. Louis U. School of Medicine, 1952.

Certificates of Registration Granted

Simon Kramer, M.R.C.S., England, 1943; L.R.C.P., London, 1943; M.B., B.S.., U. London, 1943; D.M.R., R.C.P.S., England, 1949; F.F.R., England, 1952.

Edward John Walter Alexander Ormond Bowie, B.M., B.Ch., Oxford U., 1952.

Gregor Bronstein, M.D., l'Aurore U., 1935; M.D., U. Aix-Marseilles, 1940; L.M.C.C., 1954.

Certificate of License Confirmed

Louis Paul Marier, M.D., Ottawa U., 1954; L.M.C.C., 1954.

Enabling Certificate Fee

The President inquired whether the full registration of One Hundred Dollars ($100.00) should be collected, in addition to the Documentation Fee ($25.00) and Enabling Certificate fee ($25.00), of which $20.00 is rebated, upon subsequent registration.

Motion: "THAT reconsideration be given to discontinuation of rebate of Twenty Dollars ($20.00) of the Twenty-five Dollar ($25.00) Enabling Certificate fee, to candidates who register but do not intend to practise in Manitoba." Carried.

Fee required from Hospital Employes

Motion: "THAT full registration be required from hospital employees who are earning more than Two Hundred and Seventy-five Dollars ($275.00) monthly." Carried.

Executive Committee

Winnipeg, Manitoba
November 24th, 1954

A meeting of the Executive Committee was held in the Medical Arts Club Rooms at 1.00 p.m. on Wednesday, November 24th, 1954.

Present: Dr. C. B. Stewart, Chairman, Dr. M. R. MacCharles, Dr. G. H. Hamlin, Dr. Ed. Johnson, Dr. F. P. Doyle, Dr. C. H. A. Walton, President ex-officio, and Dr. M. T. Macfarland, Registrar ex-officio.

1. Cancer Relief and Research Institute.

The President stated that the question of representation from the College to the Board of the Cancer Relief and Research Institute had been discussed at the annual meeting of Council on October 16th. He advised that at the last meeting of the Board, at which Dr. M. R. MacCharles was present as Chairman of the Medical Advisory Committee, the by-laws of the Institute were being revised. Since there is a lack of continuity in the members of the Board of Trustees, he took the opportunity to point out that the C.P. and S. had an unsatisfactory representation since the President and Registrar are ex-officio members appointed by the Act, and that the President changed annually and often had to come long distances to attend meetings. The Minister of Health stated he would bring forward an amendment to The Cancer Relief Act to provide for two members of the Council of the College to act as representatives on the Board of the Institute.

Motion: "THAT the Registrar be instructed to write to the Chairman of the Board of Trustees of the Cancer Relief and Research Institute, requesting that steps be taken to amend The Cancer Relief Act, changing the representation of this College from the present ex-officio representation, namely President and Registrar, to two members appointed by Council." Carried.

Social News

Reported by K. Borthwick-Leslie, M.D.

Greetings to all friends and confreres from our erstwhile members who are now in B.C. I hadn't time to see any of those in Vancouver, but talked to Archie Hardyment, Andy Turnbull and Lynne Gunn by phone the night before I left, and hear via Shaughnessy Golf Course that Drs. Gordon Fahrni (Sr.) and Eddie Alexander are "raring to go" — also A. M. Davidson. Couldn't find Harry Lewis, but friends had met him on the street and reported that he looks like a million in spite of a heart attack some time ago.

♦

In Victoria, by hospitality of my pal Kate Mathers, some of you remember petite blond Kate, from her days as Occupational Therapist at Psycho, had a cocktail party and on twelve hours notice enjoyed very much seeing and listening to Bill McIlmoyle (still somewhat crippled with arthritis, but a mighty important and busy Surgeon), Bobby Hunter, Harry Grieve, Johhny Cruise, Roy Fraser, Bob Whitehead, Don Revell, Mac Edmison (brother Jack couldn't come), Frank Stuart and Norm Cook. Being a Thursday only a few wives were able to come, but they certainly are all very happy, busy, healthy and much taken with Island weather, rain and all. Bobby Roberts, Matron of the Veterans' Hospital, also came and sends her greetings to all the boys of the R.C.A.M.C. She looks wonderful. So much for the B.C. crowd.

♦

Dr. James S. McGoey announces the opening of his office at 306 Medical Arts Bldg. Practice limited to General, Thoracic and Cardiovascular Surgery.

♦

Dr. and Mrs. James S. McGoey are happy to announce the arrival of Therésa Margaret Mary, March 6, 1955.

♦

Dr. Arnold G. Rogers, M.D., M.Sc. (Med.), F.R.C.P. (C.) announces his association with the Mall Medical Group of Winnipeg in the practice of Internal Medicine and Gastro Enterology.

The Manitoba Clinic wishes to announce that John Edgar McGoey, B.A., M.D. has been appointed Otorhinolaryngologist.

♦

Dan R. Bigelow, M.D., M.Sc., Orthopedic Surgery, American Board, Part 1, announces the opening of his office at 19 Medical Arts Bldg. for Orthopedic and Traumatic Surgery.

♦

Dr. and Mrs. R. E. Beamish are happy to announce the birth of Mary Anne, April 15th.

♦

Dr. and Mrs. C. D. Lees announce the arrival of John Daniel, April 18th.

♦

Dr. and Mrs. T. Krawchuk announce the birth of a son on April 17th.

♦

Dr. and Mrs. Jack Rubin are proud to announce the arrival of Jacqueline, a sister for Barbara Ruth on April 12th.

♦

Dr. and Mrs. Melville M. Brown announce the engagement of their only daughter Margaret Craig to Thomas Leonard Thomson. The wedding will take place May 14th in St. Andrews United Church.

♦

Due to my absence during March and early April, I probably have missed out much news of note. Sorry — not deliberate.

♦

P.S. — How come some of my confreres are calling some of my families, suggesting they report en masse for Salk Vaccine. Supposedly one in the profession has already given 20 and has adequate supply for all ages, non pregnant as well. I have not been able to get even **one** vial for my pregnancies.

Who **is** in control?

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1955		1954		Total	
	Feb. 27 to Mar. 26,'55	Jan. 30 to Feb. 26,'55	Feb. 21 to Mar. 20,'54	Jan. 24 to Feb. 20,'54	Jan. 2 to Mar. 26,'55	Jan. 1 to Mar. 20,'54
Anterior Poliomyelitis	1	1	5	8	2	19
Chickenpox	172	203	182	187	532	561
Diphtheria	0	0	0	0	1	0
Diarrhoea and Enteritis, under 1 year	1	6	14	14	9	36
Diphtheria Carriers	0	2	0	0	2	0
Dysentery—Amoebic	0	0	0	0	0	0
Dysentery—Bacillary	1	0	1	3	2	4
Erysipelas	1	2	3	3	3	7
Encephalitis	0	0	0	0	0	0
Influenza	12	2	7	7	20	18
Measles	494	606	155	81	1351	293
Measles—German	27	8	1	5	39	6
Meningococcal Meningitis	2	3	0	2	7	2
Mumps	172	214	197	150	500	409
Ophthalmia Neonatorum	0	1	0	0	1	0
Puerperal Fever	0	0	0	0	0	0
Scarlet Fever	19	30	63	70	72	194
Septic Sore Throat	1	6	11	3	7	19
Smallpox	0	0	0	0	0	0
Tetanus	0	0	0	0	0	0
Trachoma	0	0	0	0	0	0
Tuberculosis	42	36	51	29	94	87
Typhoid Fever	0	0	0	2	0	2
Typhoid Paratyphoid	0	0	0	0	0	0
Typhoid Carriers	0	0	0	0	0	0
Undulant Fever	0	0	1	0	0	1
Whooping Cough	78	92	11	5	233	20
Gonorrhoea	60	93	100	95	233	296
Syphilis	15	19	7	8	38	18
Jaundice Infectious	20	45	28	32	88	74

Four-week Period February 27th to March 26th, 1955

DISEASES (White Cases Only)	*828,000 Manitoba	*861,000 Saskatchewan	*2,825,000 Ontario	*2,952,000 Minnesota
*Approximate population.				
Actinomycosis	....	1	....	....
Anterior Poliomyelitis	1	....	1	5
Chickenpox	172	1	2138	....
Diarrhoea and Enteritis, Under 1 Year	1	12	....	....
Diphtheria	....	1	3	1
Diphtheria Carriers	....	....	....	....
Dysentery—Amoebic	....	....	....	2
Dysentery—Bacillary	1	12	18	7
Encephalitis, Infectious	....	....	....	....
Erysipelas	1	....	1	....
Influenza	12	440	1629	....
Jaundice, Infectious	20	91	76	182
Measles	494	36	2494	1930
German Measles	27		3315	....
Meningitis Meningococcus	2	5	7	9
Mumps	172	2	1767	....
Ophthal. Neonat.	....	....	....	....
Puerperal Fever	....	....	....	....
Scarlet Fever	19	16	281	119
Septic Sore Throat	1	43	5	91
Smallpox	....	....	....	....
Tetanus	....	....	....	....
Trachoma	....	....	....	....
Tuberculosis	42	35	105	142
Tularemia	....	....	....	....
Typhoid Fever	....	....	2	....
Typh. Para-Typhoid	....	....	....	....
Typhoid Carriers	....	....	....	....
Undulant Fever	....	1	2	17
Whooping Cough	78	17	596	50
Gonorrhoea	60	....	§93	....
Syphilis	15	....	§34	....

§These figures only for two weeks.

DEATHS FROM REPORTABLE DISEASES
March, 1955

Urban—Cancer, 71; Influenza, 1; Pneumonia, Lobar (490), 5; Pneumonia (other forms), 13; Syphilis, 2; Tuberculosis, 4; Whooping Cough, 1; Septicaemia and pyaemia, 2; Diarrhoea and Enteritis, 1. Other deaths under 1 year, 28. Other deaths over 1 year, 195. Stillbirths, 13. Total, 336.

Rural—Cancer, 25; Influenza, 5; Pneumonia, Lobar (490), 4; Pneumonia (other forms), 15; Tuberculosis, 5; Meningococcal Infections, 1. Other deaths under 1 year, 21. Other deaths over 1 year, 171. Stillbirths, 13. Total, 260.

Indians—Influenza, 1; Pneumonia (other forms), 3; Tuberculosis, 1*. Other deaths under 1 year, 4. Other deaths over 1 year, 4. Total, 13.
*2 Whites on Indian Reserve.

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Anterior Poliomyelitis—Since March 26th a second case of poliomyelitis has been reported but without paralysis.

Vaccine — Dr. Francis, Jr., and Dr. Jonas Salk have now issued their reports and our vaccination is going ahead as of the week commencing April 18th. Second doses will be given **four** weeks later and at that time Grade II pupils will be included in the City of Winnipeg and eight year olds in the balance of the Province.

Measles—Still quite prevalent.

Influenza — There have been many cases but only a few reported.

Jaundice Infectious is quite troublesome in several areas. It is not as well reported as it should be.

Book Review

Aphasia Therapeutics. Mary C. Longerich, Ph.D. and Jean Bardeaux, Ph.D., McMillan Co., New York, 1954, 185 pages. Price $3.75.

This monograph embodies more than the treatment of aphasia. There are more pages devoted to a detailed description of various types of aphasia and their examination, than there is to management. Accompanying the description of each type of aphasia, is the physiological background, which is a statement as to the supposed site of the lesion, causing the particular type of aphasia.

The instructions for carrying out therapy are given in great detail. These are lucid and easy to follow. The same can be said for the rest of the text. It is a well and simply written treatise with no ambiguity. All the complicated words, which are used to describe the aphasias, such as paragammatism and autotopognasia are clearly defined.

As is to be expected, the book is weak in the strictly clinical neurological aspect, but this forms an insignificant part of the whole, and does not detract from its overall usefulness. J. M.

Motion Picture Films

A completely revised Fourth Edition of "Professional Films" is now in compilation. (The frequency and number of future insert pages necessary to assure a comprehensive index that is continuously current over a period of years will be determined by the volume of forthcoming productions.) It will include new sections providing biographical data on authors, and information on the audio-visual activities of medical schools, dental schools and post-graduate teaching centers.

Over 28,000 copies of previous Editions are in use by medical and dental schools, Program Chairmen of State and specialty societies, and others here and abroad. AIM provides this valuable audio-visual information to the profession-at-large, without profit, as one of its contributions toward elevating the standards of medical and dental services by expediting the disseminating of the professional knowledge.

You are urged to directly assist by (1) informing film authors of this announcement so that they can write for questionnaires, or (2) providing the film title and full name and address of any film author. Write to the Academy-International of Medicine, 601 Louisiana Street, Lawrence, Kansas.

Tenth Annual Schering Award

Competition Opens for Nation's Medical Students

The tenth annual Schering Award competition for medical students has opened, it was announced by Robert W. Burlew, M.D., chairman of the award committee. Its aim is to encourage medical writing and exploration of current research literature.

Titles of three subjects on which American and Canadian students are invited to submit papers this year are: Current Concepts in the Management of Osteoporosis; Prevention and Treatment of Blood Transfusion Reactions; and Recent Trends in the Clinical Use of Adrenocortical Steroids.

Both a $500 first prize and a second one of $250 will be awarded for each of the three subjects. Outstanding authorities in each field will select the winners. Special recognition in the form of a professional gift will be given each student submitting a meritorious paper.

Deadline for entry forms specifying the choice of title is July 1st. Manuscripts should be mailed not later than October 1st. Students may compete individually or cooperatively in teams.

The Schering Award Committee brings outstanding papers to the attention of appropriate professional journals. Several papers by award winners of recent years have already been published in journals.

Dr. Burlew said that publication of these papers demonstrates the high level of research and medical writing attained by many participants in this traditional competition.

Information and instructions for the award competition are available from the Schering Award Committee, 60 Orange Street, Bloomfield, N.J.

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